WEST Search History

DATE: Friday, November [illegible], [illegible]

Set Name side by side	Query	Hit Count	Set Name result set
DB= USPT,PGPB,JPAB,EPAB,DWPI; PLUR=YES; OP=ADJ			
L14	L2 and L12	2	L14
L13	L1 and L12	0	L13
L12	GGGACTTTCC	64	L12
L11	L2 and L9	184	L11
L10	L1 and L9	101	L10
L9	binding sites	37632	L9
L8	L6 and L2	0	L8
L7	L6 and L1	1	L7
L6	L3 and L5	64	L6
L5	dendritic cell	3007	L5
L4	ribozym$3	8755	L4
L3	tolerogen$3	395	L3
L2	nf kappa b	424	L2
L1	nuclear factor kappa b	211	L1

END OF SEARCH HISTORY

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      (FILE 'HOME' ENTERED AT 11:34:48 ON 21 NOV 2002)

      FILE 'BIOSIS, MEDLINE, CAPLUS, EMBASE' ENTERED AT 11:34:[illegible] ON 21 NOV 2002
L1         25547 NUCLEAR FACTOR KAPPA B
L2         41469 NF KAPPA B
L3          4421 TOLEROGEN?
L4         15153 RIBOZYM?
L5        183175 OLIGONUCLEOTIDE
L6         77082 ANTISENSE
L7           628 L3 AND L4
L8             1 L7 AND L5
L9           [illegible] L6 AND L5
L10            [illegible]
L11                                  [illegible] PHOSPHO?
L12            [illegible] AND L5
L13            [illegible]
L14            [illegible] AND L5 AND L6
L15            [illegible] AND L5
L16          [illegible] ENDOTOXIN? TOLI
L17        [illegible] TOLERIZ? CELL
L18            [illegible] AND L5 AND L17
L19            11 DUP REM L16 (1 DUPLICATE REMOVED)
L20            21 L2 AND L5 AND L17
L21            15 DUP REM L20 (6 DUPLICATES REMOVED)

      FILE 'BIOSIS, MEDLINE, CAPLUS, EMBASE' ENTERED AT 11:[illegible] ON 21 NOV 2002
L22             7 L17 AND L3 AND L1
L23             9 DUP REM L12 (0 DUPLICATES REMOVED)
L24            19 L17 AND L1 AND L5
L25             6 DUP REM L24 (4 DUPLICATES REMOVED)
L26            [illegible] L17 AND L1 AND L3
L27            [illegible] L17 AND [illegible] AND L3

      FILE 'BIOSIS, MEDLINE, CAPLUS, EMBASE' ENTERED AT [illegible] ON 21 NOV 2002

      FILE 'REGISTRY' [illegible] AT [illegible] ON 21 NOV 2002
L28        [illegible] [illegible] L5

      FILE 'CAPLUS' ENTERED AT [illegible] ON 21 NOV 2002
L29            [illegible] L28 AND L1 AND L3
L30        [illegible] [illegible] L29
L31            [illegible] L1 AND L3
L32            814 L2 AND L29
L33              6 L31 AND L17
L34              6 DUP REM L33 (0 DUPLICATES REMOVED)
L35              8 L32 AND L17
L36              8 DUP REM L35 (0 DUPLICATES REMOVED)

=>
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L34 ANSWER 1 OF 4 [illegible] COPYRIGHT 2004 ACS
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: Selective expression of type I IFN genes in human **dendritic cells** infected with Mycobacterium tuberculosis
AUTHOR(S): Remoli, Maria Elena; Giacomini, Elena; Lutfalla, Georges; Dondi, Elisabetta; Orefici, Graziella; Battistini, Angela; Uze, Gilles; Pellegrini, Sandra; Coccia, Eliana M.
CORPORATE SOURCE: Laboratories of Immunology, Istituto Superi[illegible] di Sanita, Rome, I-[illegible], Italy
SOURCE: Journal of Immunology ([illegible]), [illegible], 3[illegible]-374 [illegible]: [illegible]; [illegible]: [illegible]
PUBLISHER: American Association of Immunologists
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Type I IFN [illegible] different aspects of the immune response, inducing a cell-mediated immunity. We have recently shown that the infection of **dendritic cells** [illegible] with Mycobacterium tuberculosis (Mtb) induces IFN-.alpha.. In this work we have monitored a rapid induction of IFN-.beta. followed by the delayed prodn. of the IFN-.alpha.1 and/or -.alpha.13 subtypes. The Mtb infection rapidly activates the NF-.kappa.B complex and stimulates the phosphorylation of IFN regulatory factor (IRF)-3, events known to induce IFN-.beta. expression in viral infection. In turn, the autocrine prodn. of IFN-.beta. induces the IFN-stimulated genes that contain **binding sites** for activated STATs in their promoters. Among the IFN-stimulated genes induced in DC through STAT activation are IRF-1 and IRF-7. The expression of IRF-1 appears to be dependent on the sequential activation of NF-.kappa.B and STAT-1. Once expressed, IRF-1 may further stimulate the transcription of IFN-.beta.. Induction of IRF-7 is also regulated at the transcriptional level through the binding of phosphorylated STAT-1 and STAT-2, forming the IFN-stimulated gene factor 3 complex. In turn, the IRF-1 and IRF-7 proteins are required for the delayed [illegible] IFN-.alpha. genes. Although tentative, our results strongly support the [illegible] of multiple events in Mtb-infected DC. Upon Mtb [illegible], constitutively expressed NF-.kappa.B and IRF-3 are activated and [illegible] to the rapid IFN-.beta. expression. In turn, IFN-.beta.-induced IRF-1 and IRF-7 may cooperate toward induction of IFN-.alpha. genes if Mtb infection persists and these factors are activated.
REFERENCE COUNT: [illegible] THERE ARE [illegible] CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L34 ANSWER 2 OF 4 [illegible] COPYRIGHT 2004 ACS
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: 139:[illegible]
TITLE: The use of tolerogenic **dendritic cells** for enhancing tolerogenicity in a host and methods for making the same
INVENTOR(S): Robbins, Paul D.; Lu, Lina; Giannakis, Nick
PATENT ASSIGNEE(S): University of [illegible] Pittsburgh of the Commonwealth System of Higher Education, USA
SOURCE: PCT Int. Appl., [illegible] pp.
[illegible]: [illegible]
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: [illegible]
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
[illegible]	A[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	A[illegible]	[illegible]	[illegible]	[illegible]

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, [illegible]
RW: [illegible] ZW, AT, BE, CH, CY,
DE, DK, [illegible], FR, [illegible], IE, IT, LU, MC, NL, PT, SE, TR, BF,
BJ, CF, [illegible], CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
[illegible] US 2001-844915 20010427

PRIORITY APPLN. INFO.: US 2000-200479P P 20000428

AB The present invention relates to a tolerogenic mammalian **dendritic cells** (DCs) and methods for the prodn. of the tolerogenic DCs. In addn., the present invention provides a method for enhancing tolerogenicity in a host comprising administering the tolerogenic mammalian DCs of the present invention to the host. The tolerogenic DCs of the present invention comprise an oligodeoxyribonucleotide (ODN) which has one or more NF-.kappa.B **binding sites**. The tolerogenic DCs of the present invention may further comprise a viral vector, and preferably an adenoviral vector, which does not affect the tolerogenicity of the tolerogenic DCs when present therein. Enhanced tolerogenicity in a host is useful for prolonging foreign graft survival and for treating inflammatory related diseases, such as autoimmune diseases.

L34 ANSWER 3 OF [illegible] COPYRIGHT [illegible]

ACCESSION NUMBER: [illegible]

DOCUMENT NUMBER: [illegible]

TITLE: [illegible] cardiac allograft survival using **dendritic cells** treated with NF-.kappa.B decoy oligodeoxyribonucleotides

AUTHOR(S): Giannoukakis, Nick; Bonham, C. Andrew; Qian, Shiguang; Zhou, Zhongyou; Peng, Lansha; Harnaha, Jo; Li, Wei; Thomson, Angus W.; Fung, John J.; Robbins, Paul D.; Lu, Lina

CORPORATE SOURCE: Department of Molecular Genetics and Biochemistry, University of Pittsburgh, Pittsburgh, PA, 15261, USA

SOURCE: Molecular Therapy (2000), 1(5, Pt. 1), 430-437
CODEN: MTOHCK; ISSN: 1525-0016

PUBLISHER: Academic Press

DOCUMENT TYPE: Journal

LANGUAGE: English

AB **Dendritic cells** [illegible] classically prime immune responses [illegible] antigen-specific [illegible] costimulatory molecules (CD40, [illegible] with their capacity to [illegible] these molecules is associated with NF-.kappa.B [illegible] their genes. DC [illegible] with impaired NF-.kappa.B-dependent transcription [illegible] as well as NF-.kappa.B translocation to the nucleus. In this report, we demonstrate that double-stranded oligodeoxyribonucleotides [illegible] **binding sites** for NF-.kappa.B (NF-.kappa.B ODN) are efficiently incorporated by bone marrow-derived [illegible] specifically inhibit NF-.kappa.B-dependent transcription of a reporter gene. Moreover, exposure of DC to the oligonucleotide decoys inhibited lipopolysaccharide (LPS)-induced nitric oxide prodn., a marker of DC maturation. Treatment of bone marrow-derived DC progenitors with NF-.kappa.B ODN [illegible] cell-surface expression of costimulatory molecules, without interfering with MHC class I or class II expression. Furthermore, NF-.kappa.B [illegible] induced [illegible] cultures, [illegible] Finally, [illegible] NF-.kappa.B [illegible]

significant prolongation of allograft survival in the absence of immunosuppression. Specific interference with NF-.kappa.B and other transcriptional pathways involved in immune stimulation in DC using DN decoy approaches could be one means to promote tolerance induction in organ transplantation. (c) 2000 Academic Press.

REFERENCE COUNT: 74 THERE ARE 74 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L84 ANSWER [illegible]
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] **dendritic cell** maturation through the inhibition of **nuclear factor-. kappa.B** activation in hemopoietic progenitor cells

AUTHOR(S): [illegible]yama, Tsuneharu; Ran, Sophia; Ishida, Tadao; Nadaf, Sirena; Kerr, Lawrence; Carbone, David P.; Gabrilovich, Dmitry I.

CORPORATE SOURCE: The Vanderbilt Cancer Center and Departments of Medicine and Microbiology and Immunology, Vanderbilt University School of Medicine, Nashville, TN, 37232, USA

SOURCE: Journal of Immunology (1998), 160(3), 1224-1232
CODEN: JOIMA3; ISSN: 0022-1767

PUBLISHER: American Association of Immunologists
DOCUMENT TYPE: Journal
LANGUAGE: English

AB [illegible] **dendritic cells** [illegible] during the [illegible] **binding sites** for VEGF decreased [illegible] with decreased levels of mRNA for Flt-1. VEGF significantly inhibited **nuclear factor-. kappa.B** (NF-.kappa.B)-dependent activation of reporter gene transcription during the first 24 h in culture. The presence of VEGF significantly decreased the specific DNA binding of NF-.kappa.B as early as 30 min after incubation with TNF-.alpha.. This was followed on days 7 to 10 by decreases in the mRNA for RelB and c-Rel, two subunits of NF-.kappa.B. Blockade of NF-.kappa.B activity in HPC at early stages of differentiation with an adenovirus expressing a dominant I.kappa.B inhibitor of NF-.kappa.B reproduced the pattern of effects induced with VEGF. Thus, NF-.kappa.B plays an important role in maturation of HPC to DC, and VEGF activation of the Flt-1 receptor [illegible] activation of NF-.kappa.B in this system. [illegible] NF-.kappa.B activation in HPC by tumor-derived factors may [illegible] a mechanism by which [illegible] the ability of the immune system [illegible].

L84 ANSWER [illegible]
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] promoter-mediated [illegible] NF-.kappa.B and primer [illegible] for IL-12 p40 gene

AUTHOR(S): [illegible]; Bellarosa, Maria L.; Bianchi, [illegible]; [illegible]; Ayo [illegible], Emilia; Piconetti, Maria L.; [illegible], [illegible]

CORPORATE SOURCE: [illegible] Experimental [illegible], University [illegible], [illegible], [illegible], Italy

SOURCE: Immunity [illegible], [illegible], [illegible]

[illegible]; [illegible]

[illegible]:
[illegible]:
[illegible]:
AB The [illegible] receptor by fresh **dendritic cells** [illegible]. Using RT-PCR, RNAse protection, and electrophoretic mobility shift assay anal., they found that DC possess an IL-12 receptor with .beta.1 subunit (downstream box 1)-related differences from that on T cells. IL-12 signaling through this receptor involved members of the NF-.kappa.B but not STAT family. The unique properties of the IL-12 receptor in DC, characterized by a single class of **binding sites** with a Kd of about 300 pM, may underlie rather unique effects, such as IFN.gamma.-independent augmentation of class II antigen expression and priming for IFN-induced prodn. of IL-12.

REFERENCE COUNT: 48 THERE ARE 48+ TIMES REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS ARE AVAILABLE IN THE RE FORMAT

134 ANSWER [illegible]

ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] transcript by CD40 [illegible] of nuclear **factor-.kappa.B**

AUTHOR(S): [illegible]; Nagase, Hisashi; Ishida, Takaomi; Inoue, Junichiro; Mariuchi, Hideo

CORPORATE SOURCE: Department Allergology, Institute Medical Science, University Tokyo, Tokyo, 108, Japan

SOURCE: European Journal of Immunology (1997), 27(12), 3461-3471
CODEN: EJIMAF; ISSN: 0014-2980

PUBLISHER: Wiley-VCH Verlag GmbH
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Interleukin-12 is produced in response to infection with bacteria or parasites or to bacterial constituents such as lipopolysaccharide (LPS) in monocytes/macrophages and **dendritic cells**, and is generated by the interaction between activated T cells and antigen-presenting cells via CD40-CD40 ligand (CD40L). Transcriptional analysis of the [illegible] of many cytokine genes, including those such as IL-6 and TNF, revealed that the induction was characteristic of a primary response gene. Here, I and a human [illegible], THP-1, Raji cells, stimulated by an [illegible] with CD40L or by transfection with a CD40L expression [illegible], enhanced p40 mRNA expression. Gel shift assays using the p40 promoter revealed potential nuclear factor NF-.kappa.B **binding sites** between -1100 and -1000. Gel mobility shift assay revealed that the putative NF-.kappa.B binding sequence which is located around 120 bp upstream of the transcription initiation site in murine and human p40 genes formed an NF-.kappa.B complex with nuclear ext. from Daudi cells stimulated by CD40 ligation. Moreover, transfection of Daudi cells with the polymd. NF-.kappa.B binding sequence fused to a thymidine kinase/chloramphenicol acetyltransferase (CAT) expression plasmid greatly induced CAT activity, but transfection with the polymd. mutated NF-.kappa.B binding sequence did not. These results indicate that the NF-.kappa.B binding [illegible] of the p40 promoter [illegible] transcription [illegible] of NF-.kappa.B.

L36 ANSWER 1 OF 7 [illegible] COPYRIGHT 2004 ACS

ACCESSION NUMBER: 2002:712316 CAPLUS

TITLE: Marked prolongation of cardiac allograft survival by **dendritic cells** genetically engineered with NF-.kappa.B [illegible]

AUTHOR(S): [illegible]

CORPORATE SOURCE: [illegible] Thomas E. Starzl Transplantation Institute, University of Pittsburgh [illegible], University of Pittsburgh, Pittsburgh, PA, 15213, USA

SOURCE: Journal of Immunology (2002), 169(6), 3382-3391
CODEN: JOIMA3; ISSN: 0022-1767

PUBLISHER: American Association of Immunologists

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Bone marrow-derived **dendritic cells** (DCs) can be genetically engineered using adenoviral (Ad) vectors to express immunosuppressive mols. that promote T cell unresponsiveness. The success of these DCs for therapy of allograft rejection has been limited in part by the potential of the adenovirus to promote DC maturation and the inherent ability of the DC to undergo maturation following in vivo administration. DC maturation occurs via NF-.kappa.B-dependent mechanisms, which can be blocked by double-stranded "decoy" oligodeoxyribonucleotides containing **binding sites** for NF-.kappa.B. [illegible] NF-.kappa.B [illegible] DCs exhibit markedly impaired allostimulatory activity and promote apoptosis of activated T cells. Furthermore, administration of Ad CTLA4-Ig ODN-treated donor DCs (C57BL10; B1 [H-2b], before transplant significantly prolongs MHC-mismatched (BALB/c; CBA [H-2d]) vascularized heart allograft survival, with long-term (>100 days) donor-specific graft survival in 40% of recipients. The mechanisms responsible for DC tolerogenicity, which may involve activation-induced apoptosis of alloreactive T cells, do not lead to generation of nonspecific immunosuppressive responses. Use of NF-.kappa.B antisense decoys in conjunction with Ad encoding a potent costimulation blocking agent offers promise for therapy of allograft rejection or autoimmune disease with minimal systemic immunosuppression.

REFERENCE COUNT: [illegible] THERE ARE [illegible] OTHER REFERENCES AVAILABLE IN THE RE FORMAT. [illegible] ALL CITATIONS AVAILABLE IN THE RE FORMAT

L36 ANSWER [illegible] [illegible]

ACCESSION NUMBER: [illegible]

DOCUMENT NUMBER: [illegible]

TITLE: [illegible] dendritic cells [illegible]

AUTHOR(S): [illegible]

CORPORATE SOURCE: [illegible]

SOURCE: [illegible]

PUBLISHER: [illegible]

ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible]
LANGUAGE: [illegible]
AB [illegible] **dendritic cells** [illegible] Mycobacterium tuberculosis [illegible] IFN-.alpha.. [illegible] we have monitored a rapid induction of IFN-.alpha.1 followed by the delayed prodn. of the IFN-.alpha.1 and -.alpha.2 subtypes. The Mtb infection rapidly activates the **NF-.kappa.B** [illegible] phosphorylation of IRF regulatory factor (IRF)-3, events known to induce IFN-.beta. expression in viral infection. In turn, the autocrine prodn. of IFN-.beta. induces the IFN-stimulated genes that contain **binding sites** for activated STATs in their promoters. Among the IFN-stimulated genes induced in DC through STAT activation are IRF-1 and IRF-7. The expression of IRF-1 appears to be dependent on the sequential activation of **NF-.kappa.B** and STAT-1. Once expressed, IRF-1 may further stimulate the transcription of IFN-.beta.. Induction of IRF-7 is also dependent on the transcriptional level through the binding of phosphorylated STAT-1 and STAT-2, forming the IFN-stimulated gene factor-3 complex. In turn, the IRF-1 and IRF-7 expression appears to be regulated at the autocrine induction of the IFN-.alpha./.beta. genes. Although correlative, our results strongly support the existence of a unique set of events in Mtb-infected DC. Upon [illegible] **NF-.kappa.B** and IRF-3 activation likely contribute to the rapid IFN-.beta. expression. In turn, IFN-.beta.-induced IRF-1 and IRF-7 may participate in maximal induction of IFN-.alpha.1/13 if infection persists and these factors are activated.

REFERENCE COUNT: 70 THERE ARE 70 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L36 ANSWER 3 OF 8 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:616872 CAPLUS
DOCUMENT NUMBER: 135:355016
TITLE: The use of tolerogenic **dendritic cells** for enhancing tolerogenicity in a host and methods for making the same
INVENTOR(S): Robbins, Paul D.; [illegible]
PATENT ASSIGNEE(S): [illegible]
SOURCE: [illegible]
DOCUMENT TYPE: [illegible]
LANGUAGE: [illegible]
FAMILY ACC. NUM. COUNT: [illegible]
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]

DESIGNATED STATES: [illegible]

AB [illegible] **dendritic cells** [illegible]

... tolerogenicity in vivo ... mammalian DCs of the present invention ... of the present invention comprise an oligodeoxyribonucleotide (ODN) which has one or more **NF-.kappa.B binding sites.** The tolerogenic DCs of the present invention may further comprise a viral vector, and preferably an adenoviral vector, which does not affect the tolerogenicity of the DCs ... Brief ... sterile ...

L36 ANSWER ... OF ... ON STN
ACCESSION NUMBER: ...
DOCUMENT NUMBER: ...
TITLE: Prolongation of cardiac allograft survival using **dendritic cells** treated with **NF-.kappa.B** decoy oligodeoxyribonucleotides
AUTHOR(S): Giannoukakis, Nick; Bonham, C. Andrew; Qian, Shiguang; Zhou, Zhongyou; Peng, Lansha; Harnaha, Jo; Li, Wei; Thomson, Angus W.; Fung, John J.; Robbins, Paul D.; Lu, Lina
CORPORATE SOURCE: Department of Molecular Genetics and Biochemistry, University of Pittsburgh, Pittsburgh, PA, 15261, USA
SOURCE: Molecular Therapy (2000), 1(5, Pt. 1), 430-437
CODEN: MTOHCK; ISSN: 1525-0016
PUBLISHER: Academic Press
DOCUMENT TYPE: Journal
LANGUAGE: English
AB **Dendritic cells** ... hyperalkylating ... Hyperalkylating ... CH4, CH3, ... inducing ... with their capacity to ... with **NF-.kappa.B**-dependent transcription of ... has been assocd. with impaired **NF-.kappa.B**-dependent transcription of costimulatory genes as well as **NF-.kappa.B** translocation to the nucleus. In this report, we demonstrate that double-stranded oligodeoxyribonucleotides contg. **binding sites** for **NF-.kappa.B** (**NF-.kappa.B** ODN) are efficiently incorporated by bone marrow-derived DC and specifically inhibit **NF-.kappa.B**-dependent transcription of a reporter gene. Moreover, exposure of DC to the oligonucleotide decoys inhibited lipopolysaccharide (LPS)-induced tyrosine phosphorylation, a marker of DC maturation. Treatment of bone marrow-derived DC progenitors with **NF-.kappa.B** ODN selectively suppressed the cell-surface expression of ... without ... **NF-.kappa.B** ... LPS, while ... **NF-.kappa.B** ... Finally, treatment ... **NF-.kappa.B** ... **NF-.kappa.B** ... **NF-.kappa.B** ... Thus, ... DC ... ex vivo ... transplantation. ...
REFERENCE COUNT: ... THERE ARE ... OUT OF ... REFERENCES AVAILABLE FOR THIS ... ARE ALL ... AVAILABLE IN THE RE FORMAT

L36 ANSWER [illegible]

ACCESSION NUMBER: [illegible]

DOCUMENT NUMBER: [illegible]

TITLE: [illegible] multiple tumor necrosis factor receptor-associated factors in the signaling mechanism for receptor activator of **NF-.kappa.B**, a member of the TNFR superfamily

AUTHOR(S): [illegible]; Lacey, Mark E.; Anderson, Dirk M.; Cosman, David; Dougall, William C.

CORPORATE SOURCE: Department of Molecular Biology, Immunex Corporation, Seattle, WA, 98101, USA

SOURCE: Journal of Biological Chemistry (1998), 273(51), 34120-34127
CODEN: JBCHA3; ISSN: 0021-9258

PUBLISHER: American Society for Biochemistry and Molecular Biology

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Receptor activator of **NF-.kappa.B** (RANK) is a recently identified member of the TNF receptor superfamily that is structurally related to CD40 and **dendritic cells**. Like CD40, RANK plays significant roles in the activation of **dendritic cell** function and differentiation. Here we demonstrate the interaction of RANK with tumor necrosis factor receptor-associated factors (TRAFs) 1, 2, 3, 5, and 6 both in vitro and in cells. Mapping of the structural requirements for TRAF/RANK interaction revealed multiple TRAF **binding sites** clustered in two distinct domains in the RANK cytoplasmic tail. These TRAF binding domains were shown to be functionally important for the RANK-dependent induction of **NF-.kappa.B** and c-Jun NH2-terminal kinase activities. Site-directed mutagenesis data indicated that these TRAF **binding sites** exhibited selective binding for different TRAF proteins. In particular, TRAF6 interacted with membrane-proximal determinants distinct from those binding TRAFs 1, 2, 3, and 5. When this membrane-proximal TRAF6 interaction domain was deleted, RANK-mediated **NF-.kappa.B** signaling was completely inhibited while c-Jun NH2-terminal kinase activation was partially inhibited. An NH2-terminal truncation mutant of TRAF2 inhibited RANK-mediated **NF-.kappa.B** activation, but not c-Jun NH2-terminal kinase activation, [illegible] TRAF6 in kinase-independent signaling.

REFERENCE COUNT: [illegible] THERE ARE [illegible] REFERENCES AVAILABLE FOR THIS RECORD. ALL REFERENCES ARE AVAILABLE IN THE RE FORMAT

L36 ANSWER [illegible]

ACCESSION NUMBER: [illegible]

DOCUMENT NUMBER: [illegible]

TITLE: [illegible] and tissue growth factor effects [illegible] **dendritic cell** maturation through [illegible] factor receptor activator of **NF-.kappa.B** activation in hematopoietic cells

AUTHOR(S): [illegible]; Ito, Shinichi; Ito, Shigeki; Ishida, Yasuo; Nakao, Kenya; Kerr, William G.; Tanaka, Yoichi; Takahashi, Hiroyuki

CORPORATE SOURCE: The Institute [illegible], Nagoya, [illegible]

SOURCE: [illegible]

PUBLISHER: [illegible]

DOCUMENT TYPE: [illegible]

LANGUAGE: English.

AB Vascular endothelial growth factor (VEGF), produced by almost all tumor cells, affects the ability of bone marrow progenitor cells to differentiate into functional **dendritic cells** (DC) during the early stages of their maturation. In this study we demonstrate specific binding of VEGF to HPC. This binding was additionally competed by placenta growth factor (PlGF), a ligand reportedly specific for the Flt-1 receptor. The no. of **binding sites** for VEGF increased during DC maturation in vitro and was associated with increased levels of mRNA for Flt-1. VEGF also inhibited nuclear translocation of **NF-.kappa.B**. **NF-.kappa.B** activation is required for the transcription of many genes involved in the immune response. The presence of VEGF significantly reduced the constitutive DNA binding of **NF-.kappa.B** and also decreased the induction of **NF-.kappa.B** activity by monocytes in the mRNA for both Flt-1 and **NF-.kappa.B**. Blockade of **NF-.kappa.B** activation in HPC at early stages of differentiation with an adenovirus expressing a dominant I.kappa.B inhibitor of **NF-.kappa.B** reproduced the pattern of effects obsd. with VEGF. Thus, **NF-.kappa.B** plays an important role in maturation of HPCs to DC, and VEGF activation of the Flt-1 receptor is able to block the activation of **NF-.kappa.B** in this system. Blockade of **NF-.kappa.B** activation in HPCs by tumor-derived factors may therefore be a mechanism by which tumor cells can directly down-modulate the ability of the immune system to generate effective antitumor immune responses.

L36 ANSWER 7 OF 8 CAPLUS COPYRIGHT [illegible] ACS

ACCESSION NUMBER: [illegible] CAPLUS

DOCUMENT NUMBER: [illegible]

TITLE: [illegible] **NF-.kappa.B**

AUTHOR(S): [illegible]

CORPORATE SOURCE: [illegible] Experimental Medicine, University of [illegible], [illegible], Italy

SOURCE: Immunity (1994), 1(3), 213-223

CODEN: IMMUEH; ISSN: 1074-7613

PUBLISHER: Cell Press

DOCUMENT TYPE: Journal

LANGUAGE: English.

AB The authors analyzed the expression of an IL-12 receptor by fresh **dendritic cells** (DC) and a DC line. Using RT-PCR, RNase protection, and electrophoretic mobility shift assay (EMSA), they found that DC possess an IL-12 receptor with structural properties and IL-12-related differences from that on T cells. IL-12 stimulation induced receptor [illegible] and **NF-.kappa.B** but not STAT4 binding. [illegible] **binding sites** [illegible]

REFERENCE COUNT: [illegible] THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L36 ANSWER [illegible] CAPLUS COPYRIGHT [illegible] ACS

ACCESSION NUMBER: [illegible]

DOCUMENT NUMBER: [illegible]

TITLE: [illegible] **NF-.kappa.B**

AUTHOR(S): [illegible]

[illegible]

SOURCE: [illegible]
PUBLISHER: [illegible]
DOCUMENT TYPE: [illegible]
LANGUAGE: [illegible]

AB Interleukin-12 (p40) is produced in response to infection with bacteria or parasites or their bacterial constituents such as lipopolysaccharides (LPS) in monocytes/macrophages and **dendritic cells**, and also generated by the interaction between activated T cells and antigen-presenting cells via CD40-CD40 ligand (CD40L). Transcriptional analyses of p40 were carried out only using bacterial constituents such as LPS as stimuli. The transcriptional induction of p40 by CD40 ligation was characterized in a human B lymphoblastoid cell line, Daudi, and a human acute monocytic leukemia cell line, THP-1. These cells, stimulated by an agonistic monoclonal antibody against CD40 or by transfection with a CD40 expression vector, produced p40 and showed marked p40 mRNA expression. Sequence anal. of the p40 promoter revealed potential nuclear factor '**NF-.kappa.B binding sites**' [illegible]. Electrophoretic mobility shift assay revealed that the potential **NF-.kappa.B** binding sequence [illegible] bp upstream of the transcription initiation site in murine and human p40 genes formed an **NF-.kappa.B** [illegible] with nuclear extr. from Daudi cells activated by CD40 ligation. Moreover, transfection of Daudi cells with the plasmids containing **NF-.kappa.B** binding sequence linked to a thymidine kinase/chloramphenicol acetyltransferase (CAT) reporter plasmid greatly induced CAT activity, but transfection with the polymd. mutated **NF-.kappa.B** binding sequence did not. These results suggest that the **NF-.kappa.B** binding site located around 100 bp upstream of the transcription initiation site in murine and human p40 promoter regions could be important for the p40 induction by CD40 ligation via activation of **NF-.kappa.B**.

FILE 'HCAPLUS' ENTERED AT 10:44:17 ON 11 NOV 2002

FILE 'BIOSIS, MEDLINE, CAPLUS, EMBASE' ENTERED AT 10:44:17 ON 11 NOV 2002

L1 25547 NUCLEAR FACTOR KAPPA B
L2 4421 TOLEROGEN?
L3 44966 DENDRITIC CELL
L4 15153 RIBOTYM?
L5 13 L1 AND L4
L6 4 DUP REM L5 (9 DUPLICATES REMOVED)
L7 1 L6 AND L3
L8 1 DUP REM L7 (0 DUPLICATES REMOVED)
L9 11 L1 AND L3
L10 1 L9 AND L2
L11 1 L9 AND L4

FILE 'REGISTRY' ENTERED AT 10:44:17 ON 11 NOV 2002
L13 1 14 7 AND ANTISENSE/SN

FILE 'CAPLUS' ENTERED AT 10:47:42 ON 11 NOV 2002
L14 16 L13 AND L1
L15 2 L14 AND L2
L16 16 DUP REM L14 (0 DUPLICATES REMOVED)

FILE 'BIOSIS, MEDLINE, CAPLUS, EMBASE' ENTERED AT 11:34:[illegible] ON 21 NOV 2002

L1 [illegible] [illegible]
L2 [illegible] [illegible]
L3 [illegible] [illegible]
L4 [illegible] [illegible]
L5 [illegible] [illegible]
L6 77082 ANTISENS?
L7 622 L1 AND L6
L8 1 L7 AND L3
L9 1054 L2 AND L6
L10 3 L9 AND L3
L11 [illegible] [illegible]
L12 [illegible] [illegible]
L13 [illegible] [illegible]
L14 [illegible] [illegible]
L15 [illegible] [illegible]
L16 [illegible] [illegible]
L17 [illegible] [illegible]
L18 [illegible] [illegible]
L19 [illegible] [illegible]
L20 [illegible] [illegible]
L21 [illegible] [illegible]

FILE 'BIOSIS, MEDLINE, CAPLUS, EMBASE' ENTERED AT 11:49:58 ON 21 NOV 2002

L22 [illegible] [illegible]
L23 [illegible] [illegible]
L24 10 L17 AND L3 AND L2
L25 6 DUP REM L24 (4 DUPLICATES REMOVED)
L26 0 L16 AND L3 AND L1
L27 0 L16 AND L3 AND L2

=>

L19 ANSWER [illegible] ABSTRACTS INC.DUPLICATE [illegible]

ACCESSION NUMBER: [illegible]

DOCUMENT NUMBER: [illegible]

TITLE: [illegible] ERK, p[illegible] and NF-kappaB signal transduction in [illegible] of TLR[illegible], TLR4 and TLR9 gene expression induced by lipopolysaccharide in mouse **dendritic cells**.

AUTHOR(S): An, Huazhang; Yu, Yizhi; Zhang, Minghui; Xu, Hongmei; Qi, Runzi; Yan, Xiaoyi; Liu, ShuXun; Wang, Wenya; Guo, Zhenghong; Guo, Jun; Qin, Zhihai; Cao, Xuetao (1)

CORPORATE SOURCE: (1) Institute of Immunology, Second Military Medical University, 800 Xiangyin Road, Shanghai, [illegible]: caoxt@public3.sta.net.cn China

SOURCE: Immunology, (May, 2002) Vol. 106, No. 1, pp. [illegible]. http://www.blackwell-science.com/ cgilib/[illegible] ISSN: [illegible].

DOCUMENT TYPE: [illegible]

LANGUAGE: [illegible]

AB Toll-like [illegible] capable of recognizing [illegible] such as lipopolysaccharide (LPS) and [illegible] **oligonucleotides** (CpG ODN). TLR2 and TLR4 are major [illegible] for Gram-positive and Gram-negative bacterial cell wall components, respectively. TLR9 is necessary for CpG signalling. LPS or CpG ODN [illegible] immature **dendritic cells** (DC) and induce DC maturation characterized by production of cytokines, up-regulation of co-stimulatory molecules, and increased ability to activate T cells. However, little is known regarding the regulation of TLR gene expression in mouse DC. In this study, we investigated the regulation of TLR2, TLR4 and TLR9 gene expression by LPS in murine immature DC. TLR2, TLR4 and TLR9 mRNA were up-regulated following LPS stimulation. The up-regulation of TLR9 expression coincided with significantly increased production of tumour necrosis factor-alpha induced by LPS plus CpG ODN. While inhibition of extracellular signal-related kinase and NF-kappaB activation suppressed the up-regulation of the expression of TLR[illegible], TLR4 and TLR9 mRNA, inhibition of p[illegible] kinase [illegible] TLR2 and TLR4 mRNA expression [illegible] expression. These results [illegible] TLR[illegible], TLR4 and TLR[illegible] gene expression [illegible] DC. Up-regulation [illegible] overall [illegible] explain the synergy between [illegible] in the induction of cytokine production.

L19 ANSWER [illegible] BIOLOGICAL ABSTRACTS INC.

ACCESSION NUMBER: [illegible]

DOCUMENT NUMBER: [illegible]

TITLE: Expression [illegible] NFkappa-B pathway genes in **dendritic cells** [illegible] macrophages assessed by gene expression profiling.

AUTHOR(S): Faltathakis, Ioannis (1); Alcantara, Orlando (1); Boldt, David H. (1)

CORPORATE SOURCE: (1) Medicine/Hematology, University of Texas Health Science Center, San Antonio, TX USA

SOURCE: [illegible]

DOCUMENT TYPE: [illegible]

LANGUAGE: [illegible]

SUMMARY LANGUAGE: [illegible]

AB NFkappa-B/Rel transcription factors (mainly c-Rel, RelB, and p50) have been implicated in the differentiation of monocytes to either DCs or macrophages, as well as in the maturation of DCs from an immature to antigen-presenting cells. Recent studies of the expression pattern of Rel proteins and their inhibitors (IkappaBs) suggest that their activity during this differentiation process is transcriptional. To investigate differential gene expression between macrophages and DCs, we used commercially available microarrays (GEArray™ Kit), which included 4 of the NFkappa-B/Rel family genes (c-Rel, p105/p50, RelB, and c-Rel/p65) and 22 additional genes that are known to be under transcriptional control of NFkappa-B/Rel proteins. Human adherent peripheral blood monocytes were cultured with GM-CSF + IL-4 (DCs) or GM-CSF + IL-4 + TNF-alpha (mature DCs) or M-CSF (macrophages) and treated with LPS (1 microg/mL) for the last 24 hours of culture to induce maturation. Cells were harvested after 7 days, cDNA was prepared and radiolabeled with alpha-32P-dCTP, then hybridized to gene arrays containing specific gene probes. beta-actin and GAPDH or PUC18 **oligonucleotides** served as positive or negative controls, respectively. The expression of all 4 NFkappa-B/Rel family genes examined was significantly up-regulated in maturing DCs compared to macrophages. The strongest difference was observed for c-Rel. Sequential RT-PCR determinations of c-Rel, RelB, and p105 mRNAs confirmed these observations. Stimulation of macrophages with LPS resulted in induction of the same genes, but the expression of c-Rel remained higher in DCs. Among the 32 NFkappa-B/Rel target genes, 11 were consistently up-regulated in mature DCs compared to macrophages. These genes were I-kappaBalpha, MIK (serine/threonine protein kinase), ICAM-1, P-selectin, TNFR2, TNFAIP3 (tumor necrosis factor alpha-induced protein3, IL-1alpha, IL-1R1, IL-1R2, IRAK (IL-1 receptor-associated kinase), and TRAF (TNF family member-associated NFkappa-B activator). By contrast, only mip-1 (monocyte chemotactic protein) was consistently up-regulated in macrophages. Results were confirmed by the subsequent experiments. Genes whose expression did not change between the two types of cells included c-myc, VCAM-1, E-NP, NF-IL6, IL-, IL-, and IL-. We conclude that NFkappa-B/Rel family genes, especially c-Rel, are selectively induced during **dendritic cell** maturation. Moreover, this process is associated with expression of a unique subset of genes that are transcriptionally targeted by NFkappa-B/Rel factors.

L19 ANSWER 3 OF 11 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2002:54711 CAPLUS
DOCUMENT NUMBER: 136:15[illegible]
TITLE: Potential role of phosphatidylinositol 3 kinase, rather than DNA-dependent protein kinase, in dsRNA-induced immune activation
AUTHOR(S): Ishii, Ken J.; Takeshita, Fumihiko; Gursel, Ilhan; Gursel, Mayda; Conover, Jacqueline; Klinman, Dennis; Klinman, Dennis M.
CORPORATE SOURCE: [illegible]
SOURCE: [illegible]
PUBLISHER: [illegible]
DOCUMENT TYPE: [illegible]
LANGUAGE: [illegible]
AB [illegible] dsRNA stimulates a host innate immune response. Here we demonstrate that DNA-dependent protein kinase (DNA-PK) mediates dsRNA signaling. Specifically, wortmannin, an inhibitor of phosphatidylinositol 3 kinase (PI3K)-related kinases including DNA-PK, interferes with dsRNA-dependent cell activation, and DNA-PK mice fail to respond to dsRNA stimulation. Further studies [illegible]

W [illegible] DNA with [illegible] DNA-PK, thereby preventing Cp [illegible] to the NF-.kappa.B enhancer. We find that DNA-PK is not [illegible] in this process, since three strains of DNA-PK KO mice responded normally to CpG DNA. These results support a model in which CpG [illegible] to activate the TLR-9 but not DNA-PK, and suggest that a phosphoinositide-3-kinase member of the PIK-kinase family play a critical role in this activity by CpG DNA to TLR-9.

REFERENCE COUNT: 16 THERE ARE 16 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L19 ANSWER 4 OF 11 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:499249 CAPLUS
DOCUMENT NUMBER: 135:71280
TITLE: Activation and inhibition of the immune system
INVENTOR(S): Foxwell, Brian; Feldmann, Marc
PATENT ASSIGNEE(S): The Kennedy Institute of Rheumatology Trust, UK
SOURCE: PCT Int. Appl., [illegible] pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: [illegible]
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001047543	A	20010705	WO 2000-GB4925	20001222
WO 2001047543	A2	20020117		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1244466	A2	[illegible]	[illegible]	[illegible]
R: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				

PRIORITY APPLN. INFO.: [illegible]

AB A [illegible] NF-.kappa.B inhibitors, induction of an altered [illegible] NF-.kappa.B inhibitors and the inhibition and activation of immune responses by the administration of an activator or inhibitor of NF-.kappa.B is disclosed. Examples of NF-.kappa.B inhibitors include I.kappa.B.alpha., I.E, a nucleotide sequence encoding I.kappa.B.alpha., anti-sense nucleic acid encoding an NF-.kappa.B subunit, p50 or RelB, and anti-NF-.kappa.B antibodies. Examples of NF-.kappa.B inducers include NIK, MEKK, IKK2, TRAF2 and RelB. Also disclosed are methods for identifying inducers and inhibitors of NF-.kappa.B, for example adenoviral vectors.

L19 ANSWER 5 OF 11 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:472207 CAPLUS
DOCUMENT NUMBER: 135:[illegible]
TITLE: [illegible]
INVENTOR(S): [illegible]

L.; Nakahara, Scott N.; Williams, P. Mickey; Wood, William L.; Yamazaki, Taiko S.

PATENT ASSIGNEE(S): Genaera Corp., Inc., USA

SOURCE: PCT Int. Appl., 196 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO [illegible]	A1	[illegible]	WO [illegible]	[illegible]
WO [illegible]	A9	[illegible]		

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RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

WO 2000053752	A2	20000914	WO 1999-US31274	19991230
WO 2000053752	A3	20001221		

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RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

WO [illegible]	A1	[illegible]	WO [illegible]	[illegible]
WO [illegible]	A9	[illegible]		

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RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

WO [illegible]	A2	[illegible]	WO [illegible]	[illegible]
WO [illegible]	A3	[illegible]		

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

W [illegible]	A1	[illegible]	W [illegible]	[illegible]

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

AU 2001071973 A [illegible]

US 2002090681 A1 [illegible]

PRIORITY APPLN. INFO.:

[illegible]

[illegible] A 20001108
WO [illegible] W 20001108
US [illegible] A 20001220
WO [illegible] W 20010329
[illegible]

AB Proteins that are antagonists of the type of interleukin 17 and that therefore may be of use in the treatment of diseases assocd. with immune dysfunction are described. Cloning and expression vectors and host cells, fusion proteins, antibodies to the proteins, and methods of manufg. them are described. The genes for these proteins are expressed in a wide array of tissues. The proteins do not bind known interleukin 17 receptors but one of the proteins was identified as a receptor for two of the others. The proteins show interleukin 17-like activities, such as activation of NF-.kappa.B.

L19 ANSWER 6 OF 11 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:24711 CAPLUS
DOCUMENT NUMBER: 134:95802
TITLE: Antisense **oligonucleotide** [illegible] of BAFF expression
INVENTOR(S): [illegible]
PATENT ASSIGNEE(S): [illegible]
SOURCE: [illegible]
DOCUMENT TYPE: [illegible]
LANGUAGE: [illegible]
FAMILY ACC. NUM. COUNT: [illegible]
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6171862	B1	[illegible]	US [illegible]	[illegible]
WO 2001034827	A1	[illegible]	WO [illegible]	[illegible]

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

[illegible]

AB [illegible] oligonucleotides, [illegible], [illegible] of [illegible] which [illegible] BAFF [illegible] are [illegible]
nuclear factor-kappa B , [illegible]
[illegible] dendritic cell
[illegible], preferably [illegible]
oligonucleotides, [illegible] BAFF.

[illegible] **oligonucleotides** [illegible]

REFERENCE COUNT: [illegible] THERE ARE [illegible] CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L19 ANSWER [illegible] OF 11 [illegible] COPYRIGHT [illegible] ACS
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: A toll-like receptor recognizes bacterial DNA.
[Erratum to document cited in CA134:161862]
AUTHOR(S): Hemmi, Hiroaki; Takeuchi, Osamu; Kawai, Taro; Kaisho, Tsuneyasu; Sato, Shintaro; Sanjo, Hiroaki; Matsumoto, Makoto; Hoshino, Katsuaki; Wagner, Hermann; Takeda, Kiyoshi; Akira, Shizuo
CORPORATE SOURCE: Department of Host Defense, Research Institute for Microbial Diseases, Osaka University and Core Research for Evolutional Science and Technology, Osaka, [illegible], Japan
SOURCE: [illegible]
PUBLISHER: [illegible]
DOCUMENT TYPE: [illegible]
LANGUAGE: [illegible]
AB The [illegible]

L19 ANSWER [illegible] OF 11 [illegible] COPYRIGHT 2004 ACS
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: Expression of different NF-.kappa.B pathway genes in **dendritic cells** (DC) or macrophages assessed by gene expression profiling
AUTHOR(S): Baltathakis, Ioannis; Alcantara, Orlando; Boldt, David H.
CORPORATE SOURCE: Medicine/Hematology, University of Texas Health Science Center, San Antonio, TX, 78229-3900, USA
SOURCE: Journal of Cellular Biochemistry ([illegible]), [illegible], [illegible].
[illegible]
PUBLISHER: [illegible]
DOCUMENT TYPE: [illegible]
LANGUAGE: [illegible]
AB NF-.kappa.B [illegible] **dendritic cells** [illegible] **oligonucleotides** [illegible]

similarly upregulated in mature DCs compared to macrophages. The strongest difference was seen for c-rel. RT-PCR detns. of c-rel, RelB, and p105 mRNAs confirmed these observations. Among the 32 NF-.kappa.B/Rel pathway genes, 14 were upregulated in mature DCs compared to macrophages. These genes were I.kappa.B.alpha., IKK-.beta., NIK, ICAM-1, P-selectin, E-selectin, TNF-.alpha., TNFR2, TNFAIP3, IL-1.alpha., IL-1R1, IL-1RL, IRAK, and TANK. By contrast, only myd-1 [monocyte chemoattractant protein-1] was upregulated in macrophages compared to DCs. NF-.kappa.B pathway genes upregulated in DCs compared to macrophages have not previously been reported in monocytes that spontaneously differentiate into DCs or macrophages in culture. The data suggest that the expression of these genes in macrophages and DCs is tightly regulated during the differentiation of monocytes towards DCs. Moreover, the data suggest that the ability of different NF-.kappa.B/Rel dimers to activate different NF-.kappa.B signal transduction pathways in DCs and macrophages and with expression of a subset of genes in DCs that are transcriptionally regulated by NF-.kappa.B/Rel factors. The results illustrate the ability of the NF-.kappa.B pathway to respond to different stimuli by activating in a cell-specific manner unique signaling pathways and subsets of NF-.kappa.B target genes.

REFERENCE COUNT: 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L19 ANSWER 9 OF 11 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 2000:904923 CAPLUS

DOCUMENT NUMBER: 134:161862

TITLE: A Toll-like receptor recognizes bacterial DNA

AUTHOR(S): Hemmi, Hiroaki; Takeuchi, Osamu; Kawai, Taro; Kaisho, Tsuneyasu; Sato, Shintaro; Sanjo, Hiroki; Matsumoto, Makoto; Hoshino, Katsuaki; Wagner, Hermann; Takeda, Kiyoshi; Akira, Shizuo

CORPORATE SOURCE: [illegible]

SOURCE: [illegible], 408(6813), 740-745
CODEN: NATURS; ISSN: 0028-0836

PUBLISHER: Nature Publishing Group

DOCUMENT TYPE: Journal

LANGUAGE: English

AB DNA from bacteria but not vertebrates exerts immunostimulatory effects on mammalian immune cells, which depend on the presence of unmethylated CpG dinucleotides in the bacterial DNA. In contrast, mammalian DNA has a low frequency of CpG dinucleotides, and these are mostly methylated; therefore, mammalian DNA does not have immune-stimulatory activity. CpG DNA induces a strong T-helper-1-like inflammatory response. Accumulating evidence has revealed the therapeutic potential of CpG DNA as adjuvants for manipulating T-cell responses for cancer, allergy and infectious diseases. Despite the promising clin. uses, the mol. mechanism by which CpG DNA activates immune cells remains unclear. Here the authors show that cellular responses to CpG DNA are mediated by a Toll-like receptor, TLR9. TLR9-[illegible] dendritic cells. [illegible] TLR9. [illegible] CpG DNA from [illegible]

PATENT INFO: [illegible] THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L19 ANSWER 10 OF 11 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER:
DOCUMENT NUMBER:
TITLE:
dendritic cells
AUTHOR(S):
CORPORATE SOURCE: Department of Molecular Genetics and Biochemistry, University of Pittsburgh, Pittsburgh, PA, 15261, USA
SOURCE: Molecular Therapy (2000), 1(5, Pt. 1), 430-437
CODEN: MTOHCK; ISSN: 1525-0016
PUBLISHER: Academic Press
DOCUMENT TYPE: Journal
LANGUAGE: English
AB **Dendritic cells** (DC) classically promote immune responses but can be manipulated to induce antigen-specific hyporesponsiveness in vitro. The expression of costimulatory mols. (CD54, CD86, CD80) at the DC cell surface correlates with their ability to induce or suppress immune responses. Expression of these mols. is assocd. with NF-.kappa.B-dependent transcription. DC [illegible] with inducible NF-.kappa.B-dependent transcription [illegible] of NF-.kappa.B translocation to the nucleus. In this report, we demonstrate that double-stranded oligodeoxynucleotides containing consensus NF-.kappa.B NF-.kappa.B [illegible] specifically inhibited by bone marrow-derived DC [illegible] NF-.kappa.B-dependent transcription of a reporter gene. Moreover, addition of the **oligonucleotide** decoys [illegible] lipopolysaccharide (LPS)-induced nitric oxide prodn., a marker of DC maturation. Treatment of bone marrow-derived DC progenitors with NF-.kappa.B ODN selectively suppressed the cell-surface expression of costimulatory mols. without interfering with MHC class I or class II expression. Furthermore, NF-.kappa.B ODN DC induced allogeneic donor-specific hyporesponsiveness in mixed leukocyte cultures, and this was assocd. with inhibition of Th1-type cytokine prodn. Finally, infusion of NF-.kappa.B ODN-modified bone marrow-derived DC into allogeneic recipients prior to heart transplantation resulted in significant prolongation of allograft survival in the absence of immunosuppression. Specific interference with NF-.kappa.B [illegible] involved in immune stimulation [illegible] means to prom[illegible] 2000 Academic Press.
REFERENCE COUNT: [illegible]

L[illegible]
ACCESSION NUMBER:
TITLE:
oligonucleotides [illegible] nuclear factor .kappa.B [illegible]
[illegible]-mediated [illegible] hyporesponsiveness.
AUTHOR(S):
CORPORATE SOURCE:
SOURCE:
COUNTRY:
DOCUMENT TYPE:
FILE SEGMENT:
LANGUAGE:

SUMMARY LANGUAGE: English

AB Background. DNA containing the CpG motif is associated with immunomodulation of the innate immune response. Preexposure of macrophages to CpG DNA elicits a hyporesponsiveness to subsequent lipopolysaccharide (LPS) stimulation. We tested the hypothesis that this effect is due to decreased nuclear translocation of **nuclear factor** **kappa.B** (NK-.kappa.B). Methods. Murine macrophage-like RAW 264.7 cells were incubated with 1.0 .mu.g/mL CpG-containing **oligonucleotides** (CpG ODN), then [illegible] by restimulation with 1 .mu.g/mL LPS [illegible] minutes. The cells were cotransfected with an NF-.kappa.B [illegible] and a [illegible]. The [illegible] assayed for NF-.kappa.B [illegible] by [illegible], I.kappa.B-.alpha., I.kappa.B-.beta., and I.kappa.B-.epsilon. by Western [illegible], and [illegible] activity, and c-Jun NH(2)-terminal kinase, [illegible] activity assay. Results. NF-.kappa.B [illegible] activity was increased as demonstrated by luciferase activity assay in the prolonged CpG ODN pretreatment groups. Unlike endotoxin tolerance, CpG ODN pre-exposure increased cytoplasmic phospho-I.kappa.B.alpha. and did not abrogate mitogen-activated protein kinase activity. Conclusions. In macrophages, exposure to CpG DNA increases expression of the inhibitory p50 NF-.kappa.B homodimer and decreases NF-.kappa.B activity without inhibition of I.kappa.B kinases. Mitogen-activated protein kinase activity remains intact. Understanding those interactions between different toll receptor ligands may provide insight into novel therapeutic modalities.

L21 ANSWER 1 OF 1 [illegible]

ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] dendritic cells.
AUTHOR(S): [illegible]; Yu, Yiwei; Zhang, Minghui; Xu, Hongmei; Qi, Huan; Yan, Xiaoyi; Liu, Shukun; Wang, Wenya; Guo, Zhongmin; Gao, Xun; Wan, Xinhai; Cao, Xuetao (1)
CORPORATE SOURCE: (1) Institute of Immunology, Second Military Medical University, 800 Xiangyin Road, Shanghai, 200433: nacnt@public3.sta.net.cn China
SOURCE: Immunology, (May, 2002) Vol. 106, No. 1, pp. 38-45. http://www.blackwell-science.com/cgilib/jnlpage.asp?journal=imm&file=imm.htm. print. ISSN: 0019-2805.
DOCUMENT TYPE: Article
LANGUAGE: English
AB Toll-like receptors (TLR) are seminal receptors capable of recognizing pathogen-associated molecule patterns (PAMP) such as lipopolysaccharide (LPS) and CpG-containing **oligonucleotides** (CpG ODN). TLR2 and TLR4 are [illegible] cell wall components, respectively. TLR9 is necessary for CpG signalling. [illegible] **dendritic cells** [illegible] production of cytokines, up-regulated expression of co-stimulatory molecules, and increased ability to activate T cells. However, little is known regarding the regulation of TLR gene expression in DC. In this study, we investigated the regulation of TLR2, TLR4 and TLR9 gene expression by LPS in murine immature DC. TLR2, TLR4 and TLR9 mRNA were up-regulated following LPS stimulation. The up-regulation of TLR4 expression coincided with significantly increased production of tumour necrosis factor-alpha induced by LPS plus CpG ODN. While inhibition of extracellular signal-related kinase and NF-kappaB activation suppressed the up-regulation of the expression of TLR2, TLR4 and TLR9 mRNA, inhibition of p38 kinase prevented the up-regulation of TLR2 and TLR4 mRNA expression but enhanced the up-regulation of TLR9 expression. These results demonstrate that TLR2, TLR4 and TLR9 gene expression was differently regulated by LPS in murine immature DC. Up-regulation of TLR2, TLR4 and TLR9 expression by LPS might [illegible] overall responses [illegible] between [illegible] pr[illegible]

L21 ANSWER [illegible]
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] dendritic cells [illegible] expression.
AUTHOR: [illegible]
CORPORATE SOURCE: [illegible] University of Texas Health Science [illegible], Houston, Texas [illegible], USA.
CONTRACT NUMBER: [illegible]
SOURCE: JOURNAL OF [illegible] BIOCHEMISTRY, [illegible]
[illegible]
PUB. COUNTRY: [illegible]
DOCUMENT TYPE: [illegible]
LANGUAGE: [illegible]
FILE SEGMENT: [illegible]
ENTRY MONTH: [illegible]
ENTRY DATE: [illegible]

AB NF-kappaB/Rel transcription factors have been implicated in the differentiation of hematopoietic cells like **dendritic cells** (DCs) or macrophages, as well as in the maturation of DCs from antigen-processing to antigen-presenting cells. Recent studies of the expression pattern of Rel proteins and their inhibitors (IkappaBs) suggest that their regulation during this differentiation process is transcriptional. To investigate differential gene expression between macrophages and DCs, we used commercially available gene filter arrays (GEArray Kit, which included genes for NF-kappaB/Rel family genes, p105/p50, p100/p52, RelA, RelB, c-Rel, and IkappaBalpha, IkappaBbeta, IkappaBepsilon, Bcl-3, and IkappaBNS, as well as genes known to be transcriptionally regulated by NF-kappaB/Rel proteins, such as cytokines, chemokines, cell adhesion molecules, receptors, and enzymes). Monocytes were cultured with M-CSF or GM-CSF + IL-4 respectively for 6 days. In all of the experiments, macrophages were treated with LPS (100 ng/ml) for the last 48 h of culture to induce maturation. Cells were harvested after 7 days, cDNA was prepared and radiolabeled with alpha-[32P]-dCTP, then hybridized to gene arrays containing specific gene probes. Beta-actin and GAPDH or PUC18 **oligonucleotides** served as positive or negative controls, respectively. The expression of all four NF-kappaB/Rel family genes examined was significantly upregulated in maturing DCs compared to macrophages. The strongest difference was observed for c-rel. RT-PCR determinations of c-rel, RelB, and p105 mRNAs confirmed these observations. Among the 32 NF-kappaB/Rel pathway genes, 14 were upregulated in mature DCs compared to macrophages. These genes were IkappaBalpha, IKK-beta, NIK, ICAM-1, P-selectin, E-selectin, TNF-alpha, TNFR2, TNFAIP3, IL-1alpha, IL-1R1, IL-1R2, IRAK, and TANK. By contrast, only mcp-1 (monocyte chemotactic protein 1) was upregulated in macrophages compared to DCs. NF-kappaB pathway genes upregulated in DCs compared to macrophages were constitutively expressed in monocytes then selectively downregulated during macrophage but not DC differentiation. LPS did not induce expression of most of these genes in macrophages but LPS did induce upregulation of a few genes, including those for NF-kappaB/Rel family members, IkappaBalpha, and IkappaBepsilon. Thus, this differential expression pattern correlated with activation of different NF-kappaB signal transduction pathways in DCs and macrophages and with expression of a unique subset of genes that are transcriptionally targeted by NF-kappaB/Rel proteins. The results illustrate the ability of the NF-kappaB pathway to respond to differentiation stimuli by activating in a cell-specific manner unique signaling pathways and subsets of NF-kappaB target genes.

L21 ANSWER 3 OF 15 MEDLINE
ACCESSION NUMBER: 2000419045 MEDLINE
DOCUMENT NUMBER: 20394513 PubMed ID: 10
TITLE: **dendritic cells**
COMMENT:
AUTHOR:
CORPORATE SOURCE:
SOURCE:
PUB. TYPE:

LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH:
ENTRY DATE: Entered STN: [illegible]
Last Updated on STN: [illegible]
Last Updated on STN: [illegible]

AB **Dendritic cells** [illegible] hybrid cell lines [illegible]. The expression of costimulatory molecules (CD40, CD80, CD86) on the DC cell surface correlates with their capacity to induce or suppress immune responses. Expression of these molecules is associated with NF-kB-dependent transcription of their genes. DC tolerogenicity has been associated with impaired NF-kB-dependent transcription of costimulatory genes as well as NF-kB translocation to the nucleus. In this report, we demonstrate that double-stranded oligodeoxyribonucleotides containing binding sites for NF-kB (NF-kB ODN) are efficiently incorporated by bone marrow-derived DC and specifically inhibit NF-kB-dependent transcription of a reporter gene. Moreover, exposure of DC to the **oligonucleotide** decoys inhibited lipopolysaccharide (LPS)-induced nitric oxide production, a marker of DC maturation. Treatment of bone marrow-derived DC progenitors with NF-kB ODN selectively suppressed the cell-surface expression of costimulatory molecules without interfering with MHC class I or class II expression. Furthermore, NF-kB ODN [illegible] DC-mediated allogeneic T cell proliferation, and this was associated with [illegible] T cell-type cytokine production. Finally, infusion of NF-kB ODN-treated [illegible] DC into [illegible] allogeneic recipients prior to heart transplantation [illegible] significantly prolongation of allograft survival [illegible] without [illegible]. Specific interference with NF-kB activation might [illegible] pathways involved in immune stimulation in DC [illegible] DC-based therapeutic [illegible] to promote tolerance induction in clinical transplantation.

L21 ANSWER 1 OF 10 MEDLINE
ACCESSION NUMBER: 2002139184 MEDLINE
DOCUMENT NUMBER: 21864248 PubMed ID: 11773067
TITLE: Molecular mechanisms involved in CD43-mediated apoptosis of TF-1 cells. Roles of transcription Damm expression, and adhesion molecules.
AUTHOR: Cermak Lukas; Simova Darka; Pittner Alexander; Hrubisko Vaclav; Andera Ludek
CORPORATE SOURCE: [illegible]
SOURCE: [illegible]
PUB. [illegible]
[illegible]
LANGUAGE:
FILE SEGMENT:
ENTRY MONTH:
ENTRY DATE: [illegible]
[illegible]
[illegible]

AB CD43 [illegible], [illegible], a sialylated leukocyte surface glycoprotein, mediates both cell adhesion and transmits activation signals in T cells and **dendritic cells**. [illegible] anti-CD43 monoclonal antibody MEM-59 [illegible] induce apoptosis [illegible]. In this study we show that it also triggers apoptosis of the myeloid progenitor cell line TF-1. The kinetics of the MEM-59-induced apoptosis [illegible], with the first apoptotic cells appearing [illegible] the [illegible]; [illegible], [illegible]

[illegible] anti-CD3 (UCHT-3) or anti-CD99 mAb. The MEM- [illegible] of TF-1 cells was also inhibited by the [illegible], Daxx. CD43-mediated apoptosis was primarily by suppression of the DNA binding activity of the transcription factor Ap-1. DNA array screening revealed that the expression of several genes encoding apoptosis-regulating proteins, including 14-3-3 proteins and the granulocyte macrophage colony-stimulating factor (GM-CSF) receptor beta-subunit, was suppressed in TF-1 cells bound to immobilized MEM-59. The down-regulation of 14-3-3 proteins and GM-CSF receptor beta was accompanied by translocation of the proapoptotic protein Bad to the mitochondria. These results suggest that engagement of CD43 may, presumably through the repressing transcription, initiate a Bad-dependent apoptotic pathway.

L21 ANSWER 5 OF 15 MEDLINE
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] via [illegible] Toll-like receptor 2.
AUTHOR: [illegible] Geoffrey B; Brunn Gregory J; Kodaira Yuzo; Platt John L
CORPORATE SOURCE: Department of Immunology, Mayo Clinic, 2-66 Medical Sciences Building, Rochester, MN 55905, USA.
CONTRACT NUMBER: [illegible] NHLBI
HL 52397 NHLBI
SOURCE: JOURNAL OF IMMUNOLOGY, (2002 May 15) 168 (10) 5233-9. Journal code: [illegible]. ISSN: [illegible].
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: [illegible]
ENTRY DATE: [illegible]

AB [illegible] dendritic cells, but whether these receptors respond to [illegible] molecules is not known. We [illegible] dendritic cells might be [illegible] fragments of heparan sulfate proteoglycans. Dendritic cells were induced to mature in response to heparan sulfate as measured by costimulatory protein expression, morphology, and T lymphocyte stimulation, but this maturation was absent when Toll-like receptor 4 was mutated or inhibited. These findings suggest that Toll-like receptors in vertebrates may monitor tissue well-being by recognizing fragments of endogenous matrix molecules.

L21 ANSWER 6 OF 15 MEDLINE
ACCESSION NUMBER: [illegible] MEDLINE
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] dendritic cells [illegible]
AUTHOR: [illegible]
CORPORATE SOURCE: [illegible]
[illegible]
SOURCE: [illegible] Suppl [illegible] B-f:

[illegible] ISSN: [illegible].
PUB. COUNTRY: [illegible]
DOCUMENT TYPE: [illegible]; Article; [illegible] ARTICLE
[illegible]
[illegible]
LANGUAGE: [illegible]
FILE SEGMENT: [illegible]
ENTRY MONTH: [illegible]
ENTRY DATE: Entered STN: [illegible]
Last Updated on STN: [illegible]
Entered Medline: 20020213

AB **Dendritic cells** (DC) constitute a complex system of uniquely well-equipped antigen-presenting cells that initiate and regulate immune responses. Extensive recent studies have improved our understanding of DC development, differentiation, activation, and function. DC exist as distinct subsets that differ in their lineage affiliation, surface molecule expression, and biological function. These factors seem to determine the T-cell polarizing signals and type of T-cell response—T helper 1, T helper 2, or T regulatory—induced by DC [illegible]. Evidence has accumulated that DC play an important role in both central and peripheral tolerance via various mechanisms, including induction of T-cell anergy, immune deviation, T regulatory cell activity, and promotion of activated T-cell [illegible]. [illegible] the molecular basis of DC [illegible], emerging information suggests that [illegible], expression of death-inducing ligands [illegible], micro-environmental factors (in particular [illegible] suppressive cytokines), and inhibition of [illegible] proteins (e.g., nuclear factor-kappaB) can impair [illegible] potential of DC [illegible]. Manipulation of DC by control of their maturation and differentiation, or genetic engineering of these cells to express immunosuppressive molecules, offers potential for therapy of allograft rejection and autoimmune disease. In this brief overview, we outline principles and methods for generation of "tolerogenic" DC and outcomes that have been reported in experimental models. Space constraints limit literature citations.

L21 ANSWER [illegible] OF 15 MEDLINE
ACCESSION NUMBER: 2002[illegible]1821 MEDLINE
DOCUMENT NUMBER: 21900[illegible] PubMed ID: [illegible]
TITLE: [illegible] **dendritic cells** [illegible]
AUTHOR: [illegible]
CORPORATE SOURCE: [illegible], University of [illegible], Canada.
SOURCE: [illegible]
PUB. COUNTRY: [illegible]
DOCUMENT TYPE: [illegible]; Article; [illegible] ARTICLE
LANGUAGE: [illegible]
FILE SEGMENT: [illegible]
ENTRY MONTH: [illegible]
ENTRY DATE: Entered STN: [illegible]
Last Updated on STN: [illegible]
Entered Medline: [illegible]

AB We systematically investigated the [illegible] levels of **dendritic cells** [illegible] phenotype, [illegible]

CD86) and numerous cytokines and [illegible] (Flt-3L, G-CSF, IL-1alpha and -1beta, IL-6, IL-12, CCL-2, -3, -4, -5, -17, and -22, MIP-2, and CCR7) and were significantly better at inducing effector T cell responses in vivo. Furthermore, mice vaccinated with tumor peptide-pulsed mature DCs [illegible] without [illegible] with a weakly immunogenic [illegible] tumor [illegible] mature DCs [illegible] IL-1beta, IL-6, IL-12, IL-15, IL-18, Flt-3L, G-CSF, MIP-2, CCR7, [illegible] Taken together, our data [illegible] maturity [illegible].

L21 ANSWER [illegible] OF [illegible] COPYRIGHT 2004 ACS
ACCESSION NUMBER: [illegible]:649619 CAPLUS
DOCUMENT NUMBER: [illegible]:155756
TITLE: Potential role of phosphatidylinositol 3 kinase, rather than DNA-dependent protein kinase, in CpG DNA-induced immune activation
AUTHOR(S): Ishii, Ken J.; Takeshita, Fumihiko; Gursel, Ihsan; Gursel, Mayda; Conover, Jacqueline; Nussenzweig, Andre; Klinman, Dennis M.
CORPORATE SOURCE: Section of Retroviral Immunology, Division of Viral Products, Center for Biologics Evaluation and Research, Food and Drug Administration, National Institutes of Health, Bethesda, MD, 20892, USA
SOURCE: Journal of Experimental Medicine [illegible], [illegible], [illegible]-[illegible]
[illegible]; [illegible]
PUBLISHER: [illegible]
DOCUMENT TYPE: [illegible]
LANGUAGE: [illegible]
AB [illegible] DNA stimulates a strong innate immune response. [illegible] is evidence that DNA-dependent protein kinase (DNA-PK) mediates CpG stimulation. Specifically, wortmannin (an inhibitor of phosphatidylinositol 3 kinase (PI3)-kinases including DNA-PK) interferes with CpG-dependent cell activation, and DNA-PK knockout (KO) mice fail to respond to CpG stimulation. Current studies establish that wortmannin actually inhibits the uptake and colocalization of CpG DNA with toll-like receptor (TLR)-9 in endocytic vesicles, thereby preventing CpG-induced activation of the NF-.kappa.B signaling cascade. We find that DNA-PK is not [illegible] in this process, since three strains of DNA-PK KO mice responded normally to CpG DNA. These results support a model in which CpG stimulation is mediated through TLR-9 but not DNA-PK, and suggest that wortmannin-sensitive members of the PI3-kinase family play a critical role in activation by CpG DNA. [illegible].
REFERENCE COUNT: [illegible] THERE ARE [illegible] CITED REFERENCES AVAILABLE FOR THIS RE. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L21 ANSWER [illegible] OF [illegible] COPYRIGHT 2004 ACS
ACCESSION NUMBER: [illegible]:[illegible] CAPLUS
DOCUMENT NUMBER: [illegible]:[illegible]
TITLE: [illegible] in the [illegible] of the immune system
INVENTOR(S): [illegible], Brian; [illegible], Matt
PATENT ASSIGNEE(S): [illegible] Medical Research Institute of [illegible], USA
SOURCE: PCT Int. Appl., [illegible] pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO. KIND DATE APPLICATION NO. DATE

WO [illegible]	A[illegible]	[illegible]	[illegible]	[illegible]
WO [illegible]	A[illegible]	[illegible]	[illegible]	[illegible]

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RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PI [illegible]	[illegible]	[illegible]	PI [illegible]	[illegible]

R: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR

PRIORITY APPLN. INFO.: [illegible] A 19991224
[illegible] W 20001222

AB Activation of the immune response by **NF-.kappa.**
B inducers, inhibits an immune response by **NF-.**
kappa.B inhibitors, and the inhibition and activation of immune response by the administration of an activator or inhibitor of **NF-.kappa.B** is disclosed. Examples of **NF-.kappa.B** inhibitors include I.kappa.B.alpha., PSI, a nucleotide sequence encoding I.kappa.B.alpha., anti-sense nucleic acid encoding an **NF-.kappa.**
B sequence, such as Rel B, and anti-**NF-.kappa.**
B antibodies. Examples of **NF-.kappa.B** inducers include NIK, MEKK, IKK2, TFRRF2 and Rel B. Also disclosed are vectors encoding inducers and inhibitors of **NF-.kappa.**
B, for example aden viral vectors.

L21 ANSWER [illegible] OF [illegible] [illegible] COPYRIGHT [illegible] ACS
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] identified by sequence similarity and their use in treatment of immune system [illegible]
INVENTOR(S): [illegible], Alan; Philpott, Ellen; Fong, Sherman; Goddard, Audrey; Jaspars, Paul J.; Grimaldi, Christopher J.; Barney, Austin L.; Li, Hamhong; Hillan, Kenneth J.; Lukas, Daniel; Van, Lookeren Menno; Vandlen, Richard L.; Watanabe, Colin K.; Williams, P. Mickey; Wood, William I.; Tansaria, Daniel G.
PATENT ASSIGNEE(S): Genentech, Inc., USA
SOURCE: PCT Int. Appl., [illegible] pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: [illegible]
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]

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W: [illegible]

WO 2000053756 A2 20000914 WO 2000-US4341 [illegible]

WO 2000053756 A3 20010201

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WO [illegible] A2 [illegible] WO 2000-US5641 20000302

WO [illegible] A3 [illegible]

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WO 2000070050 A1 20001123 WO 2000-US13032 [illegible]

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RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE,
DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF,
CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

W[illegible]

W[illegible]

W: [illegible]

WO 2001016318 A1 [illegible] WO [illegible]

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CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU,
ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU,
LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE,
SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA,
ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE,
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CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

WO [illegible]

W: [illegible]

RW: [illegible]

WO [illegible]

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LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU,
SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN,
YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

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DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF,
BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

EP 1240325 [illegible]

B: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE,
TR, [illegible]

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GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT,
RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US,
UZ, VN, YU, ZA, ZW

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AM, AZ, BY, KG,
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IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN,
GW, ML, MR, NE, SN, TD, TG

WO [illegible]

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
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RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US,
UZ, VN, YU, ZA, ZW

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AM, AZ, BY, KG,
KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR,
IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN,
GW, ML, MR, NE, SN, TD, TG

[illegible]

[illegible]

[illegible]

[illegible]
NF-.kappa.B.

L21 ANSWER [illegible]
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: Antisense **oligonucleotide** inhibition of RANK expression
INVENTOR(S): [illegible]
PATENT ASSIGNEE(S): [illegible], Inc., USA
SOURCE: U.S., [illegible] pp.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6171860	B1	[illegible]	[illegible]	[illegible]
WO 2001034[illegible]	A1	[illegible]	[illegible]	[illegible]

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, [illegible] PL, PT, RO, RU, [illegible] TT, TZ, UA, UG, US, UZ, VN, [illegible]
RW: [illegible] ZW, AT, BE, CH, CY, [illegible] LU, MC, NL, PT, SE, BF, BJ, [illegible] GA, GN, GW, ML, MR, NE, SN, TD, TG

EP 1226424	A1	[illegible]	EP 2000-974012	20001030

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL

PRIORITY APPLN. INFO.: US 1999-435296 A 19991105
WO 2000-US29828 W 20001030

AB Antisense **oligonucleotides**, compns., and methods are provided for inhibiting the expression of RANK (receptor activator of nuclear factor-kappa B), a receptor known to enhance T-cell growth and **dendritic cell** function. The compns. comprise antisense compds., particularly antisense **oligonucleotides**, targeted to nucleic acids encoding RANK. [illegible]
oligonucleotides [illegible]

REFERENCE COUNT: [illegible] THERE ARE [illegible] CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L21 ANSWER [illegible]
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] changes in gene expression during [illegible] monocyte-derived **dendritic cells** [illegible] **oligonucleotide** microarrays and [illegible]
AUTHOR(S): [illegible]
CORPORATE SOURCE: Department of [illegible] University of Michigan, Ann Arbor, MI, [illegible], USA
SOURCE: [illegible]
PUBLISHER: [illegible]

Biology
DOCUMENT TYPE:
LANGUAGE:
AB **Dendritic cells**
RNA level. **oligonucleotide** microarrays complementary to
DCs. A
or maturation.
and include
cell adhesion, signaling, and lipid metab. Protein anal. of the same cell
populations was done using two-dimensional gel electrophoresis. A total
of 900 distinct protein spots were included, and 4 of them exhibited
quant. changes during DC differentiation and maturation. Differentially
expressed proteins were identified by mass spectrometry and found to
represent proteins with Ca2+ binding, fatty acid binding, or chaperone
activities as well as proteins involved in cell motility.
proteomic anal. provided an assessment of post-translational
modifications. The chaperone protein,
cleavage, yielding a novel form. The
oligonucleotide
microarray and
with
post-translational
processes.
REFERENCE COUNT:
FORMAT.

L21
ACCESSION NUMBER:
DOCUMENT NUMBER:
TITLE:
AUTHOR(S):
CORPORATE SOURCE:
SOURCE:
PUBLISHER:
DOCUMENT TYPE:
LANGUAGE:
AB

L21
ACCESSION NUMBER:
DOCUMENT NUMBER:
TITLE:
AUTHOR(S):
CORPORATE SOURCE:
SOURCE:

PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal
LANGUAGE: English

AB [illegible] stimulatory effects on mammalian immune cells, which depend on the presence of unmethylated CpG dinucleotides in the bacterial DNA. In contrast, mammalian DNA has a low frequency of CpG dinucleotides, and these are mostly methylated; therefore, mammalian DNA does not have immuno-stimulatory activity. CpG DNA induces a strong T-helper-1-like inflammatory response. Accumulating evidence has revealed the therapeutic potential of CpG DNA as adjuvants for vaccination strategies for cancer, allergy and infectious diseases. [illegible], the mol. mechanism by which CpG DNA activates immune cells remains unclear. Here the authors [illegible] CpG DNA is mediated by a Toll-like receptor, [illegible] TLR9-deficient (TLR9-/-) mice did not show any response to CpG DNA, [illegible] proliferation of splenocytes, inflammatory cytokine production [illegible] **dendritic cells**. TLR9-/- mice showed resistance to the lethal effect of CpG DNA without any [illegible] serum pro-inflammatory cytokine levels. The in vivo CpG-DNA-mediated T-helper type-1 response was also abolished in TLR9-/- mice. Thus, vertebrate immune systems appear to have evolved a specific Toll-like receptor that distinguishes bacterial DNA from self-DNA.

REFERENCE COUNT: 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L21 ANSWER 15 OF 15 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.

ACCESSION NUMBER: 2002330736 EMBASE

TITLE: Preexposure of murine macrophages to CpG-containing **oligonucleotides** results in nuclear factor .kappa.B p50 homodimer-associated hyporesponsiveness.

AUTHOR: Jin L.; Raymond D.P.; Crabtree T.D.; Pelletier S.J.; Rudy C.K.; Pruett T.L.; Sawyer R.G.

CORPORATE SOURCE: Dr. R.G. Sawyer, University of Virginia, [illegible], Charlottesville, VA [illegible], United States

SOURCE: [illegible]

COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: [illegible] and Pathological Anatomy
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Background. DNA containing the CpG motif is associated with immunomodulation of the innate immune response. Preexposure of macrophages to CpG DNA elicits a hyporesponsiveness to subsequent lipopolysaccharide (LPS) stimulation. We tested the hypothesis that this effect is due to decreased nuclear translocation of nuclear factor .kappa.B (NF-.kappa.B). Methods. Murine macrophage-like RAW 264.7 cells were incubated with [illegible] .mu.g/mL CpG-containing **oligonucleotides** [illegible] hours followed by restimulation with [illegible] LPS for [illegible] minutes. [illegible] cells were cotransfected with an **NF-.kappa.B** sensitive luciferase reporter construct and [illegible] plasmid. Cytoplasmic and [illegible] **NF-.kappa.B** [illegible] **NF-.kappa.B** [illegible] **NF-.kappa.B** [illegible] luciferase activity assay [illegible]. Unlike endotoxin [illegible] cytoplasmic phospho- [illegible] protein kinase activity. [illegible], exposure to CpG DNA increases

expression of the inhibitory p[illegible] NF-.kappa.B
homodimer and decreases NF-.kappa.B activity
without inhibiting p[illegible].kappa.B binding. Bcl-3 interacts with other
activity [illegible]. Bcl-3 [illegible]
[illegible]
m[illegible].

L23 ANSWER 1 OF [illegible] MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] **dendritic cells** [illegible]
[illegible] in transplantation and autoimmunity.
AUTHOR(S): [illegible]; Thomson, Angus W.
CORPORATE SOURCE: [illegible] Thomas E. Starzl Transplantation Institute, University of Pittsburgh Medical Center, 200 Lothrop Street, E1554, Biomedical Science Tower, Pittsburgh, PA, 15213: lul9@ox.upmc.edu USA
SOURCE: Transplantation (Baltimore), (January 15, 2002) Vol. 73, No. 1 Supplement, pp. S16-S22.
http://www.transplantjournal.com/. print.
ISSN: 0041-1337.
DOCUMENT TYPE: General Review
LANGUAGE: English
AB **Dendritic cells** (DC) constitute a complex system of uniquely well-equipped antigen-presenting cells that initiate and regulate immune responses. Extensive recent studies have improved our understanding of DC development, differentiation, activation, and function. DC exist as distinct subsets that differ in their lineage affiliation, surface molecule expression, and biological function. These factors seem to determine the T-cell priming stimulus and type of T cell response-T helper 1, T helper 2, or regulatory T cell-induced by DC [1]. Evidence has accumulated that DC play an important role in both central and peripheral tolerance via various mechanisms, including induction of T-cell anergy, immune deviation, T regulatory cell activity, and promotion of activated T-cell apoptosis. Although many of the details of the molecular basis of DC **tolerogenicity** have yet to be elucidated, emerging information suggests that costimulatory molecule deficiency, expression of death-inducing ligands (in particular Fas (CD95) ligand), microenvironmental factors (in particular anti-inflammatory/immunosuppressive cytokines), and inhibition of gene transcription regulatory proteins (e.g., nuclear factor-kappaB) can impart **tolerogenic** potential to DC (2). Manipulation of DC by control of their maturation and differentiation, or genetic engineering of these cells to express immunosuppressive molecules, offers potential for therapy of allograft rejection and autoimmune disease. In this brief overview, we outline principles and methods for generation of "**tolerogenic**" DC and discuss their [illegible]

L23 ANSWER [illegible] MEDLINE on STN
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] allograft survival by **dendritic cells** [illegible] engineered [illegible] NF-kappaB [illegible] a myeloid-like decoys and [illegible] CTLA4-Ig.
AUTHOR(S): [illegible]; Li, Xunsheng; Li, Miaoyan; Chen, Ping; Wang, Limin; Ma, Linlin; Burkstein, Holger; Morelli, Adrian E.; Thomson, Angus W.; Fung, John J.; Qian, Shiguang; Lu, Lina
CORPORATE SOURCE: [illegible] Thomas E. Starzl Transplantation Institute, University of Pittsburgh Medical Center, 200 Lothrop Street, Biomedical Science Tower, W1544, Pittsburgh, PA, [illegible]: lul@mox.upmc.edu USA
SOURCE: Journal of Immunology, [illegible], [illegible] Vol. [illegible], No. [illegible], pp. [illegible]. http: www. [illegible]. print. [illegible].
DOCUMENT TYPE: Article
LANGUAGE: [illegible]
AB [illegible] **dendritic cells** [illegible]

genetically modified using adenoviral (Ad) vectors to express immunosuppressive molecules that promote T cell unresponsiveness. The success of these DC for therapy of allograft rejection has been limited in part by the potential of the adenovirus to promote DC maturation and the inherent ability of the DC to undergo maturation following in vivo administration. DC maturation occurs via NF-kappaB-dependent mechanisms, which can be blocked by double-stranded "decoy" oligodeoxynucleotides (ODNs) containing binding sites for NF-kappaB. Herein, we describe the combined use of NF-kappaB ODNs and rAd vectors encoding CTLA4-Ig (rAd CTLA4-Ig) to generate stably immature murine myeloid DCs that express the potent costimulation blocking agent, human CTLA4-Ig. Unlike CTLA4-Ig-transduced wild-type DCs, exhibit markedly impaired allostimulatory ability and fail to undergo apoptosis in allogeneic recipients. In contrast, immature rAd CTLA4-Ig NF-treated donor DCs [illegible] ; NF-kappaB-decoy transplant significantly prolongs MHC-mismatched [illegible] ; NF-kappaB-decoy treatment of rAd CTLA4-Ig DCs further enhances heart allograft survival, with long-term [illegible] graft survival in 40% of recipients. In vivo studies responsible for DC **tolerogenicity**, which may [illegible] host-mediated apoptosis of alloreactive T cells, do not lead to [illegible] of intragraft Th cytokine responses. Use of NF-kappaB antisense oligos in conjunction with rAd encoding a potent costimulation blocking agent offers promise for therapy of allograft rejection or autoimmune disease with minimization of systemic immunosuppression.

L23 ANSWER 3 OF 5 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:816872 CAPLUS
DOCUMENT NUMBER: 135:355016
TITLE: The use of **tolerogenic dendritic cells** for enhancing **tolerogenicity** in a host and methods for making the same
INVENTOR(S): Robbins, Paul D.; Lu, Lina; Bianchi Rabbis, Mirk
PATENT ASSIGNEE(S): University of Pittsburgh of the Commonwealth System of Higher Education, USA
SOURCE: PCT Int. Appl., 44 pp.
[illegible]
DOCUMENT TYPE: [illegible]
LANGUAGE: [illegible]
FAMILY ACC. NUM. [illegible]:
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001083713	A	20011108	WO 2001-US13661	20010427
WO 2001083713	A	20020314		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

[illegible]
PRIORITY APPLN. INFO.: [illegible]
AB The present invention [illegible] **tolerogenic** [illegible] **dendritic cells** [illegible] **tolerogenic** [illegible], the present invention [illegible] **tolerogenicity** [illegible] administration of **tolerogenic** [illegible] the present invention [illegible] **tolerogenic** [illegible] the present invention [illegible] NF-kappaB [illegible] The **tolerogenic** [illegible] the present invention [illegible], and preferably [illegible] viral [illegible], which [illegible] **tolerogenicity** of

the **tolerogenic** [illegible]. Evaluation of **tolerogenicity** [illegible] survival [illegible], [illegible] allograft [illegible].

L23 [illegible]
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] mRNA microarray profiles of **tolerogenic dendritic cells**
AUTHOR(S): Dhodapkar-Pena Cortesini, N.; Fiamma, F.; Ho, E.; Marcantelli, R.; LeMoult, J.; Dalla-Favera, R.; Cortesini, R.
CORPORATE SOURCE: Department of Pathology, Columbia University, New York, NY, USA
SOURCE: Human Immunology (2001), 62(10), 1065-1072
CODEN: HUIMDQ; ISSN: 0198-8859
PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB **Dendritic cells** are crucial to the activation as well as suppression of the immune response. Previous reports have illustrated that ABC [illegible] with antigen-pulsed T suppressor cells may induce **tolerogenic**, [illegible] target cells. To characterize the molecular changes [illegible] **tolerogenic** APC, the mRNA profile of KG-1 **dendritic cells** [illegible] by allogeneic T helper and T suppressor cells were analyzed. This study now provides evidence that immature **dendritic cells** stimulated by T suppressor cells differentiate into mature **dendritic cells** with a distinct phenotype. The identification of Ts induced pathways of **dendritic cell** differentiation is critical to the development of [illegible] therapeutic strategies.
REFERENCE COUNT: 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L23 ANSWER 5 OF 5 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2002:199192 CAPLUS
DOCUMENT NUMBER: 137:41416
TITLE: Prolongation of cardiac allograft survival using **dendritic cells** treated with NF-.kappa.B decoy oligodeoxyribonucleotides
AUTHOR(S): [illegible]
CORPORATE SOURCE: [illegible]
SOURCE: [illegible]
PUBLISHER: [illegible]
DOCUMENT TYPE: [illegible]
LANGUAGE: English
AB **Dendritic cells** [illegible] essentially play a role in immune responses for [illegible] induction of Ag-specific hyporesponsiveness to [illegible]. The expression of stimulatory mols. (CD40, CD80, CD86 and MHC class II) [illegible] with their capacity to induce or suppress immune responses. Experiments [illegible] with NF-.kappa.B-dependent transcription of [illegible] **tolerogenicity** has been [illegible] with impaired NF-.kappa.B-dependent transcription of costimulatory mols as well as NF-.kappa.B translocation to the nucleus. In this report, we demonstrate that double-stranded [illegible] NF-.kappa.B NF-.kappa.B [illegible] NF-.kappa.B [illegible]

transcription [illegible]. Moreover, exposure of DC to the cell-permeable peptide inhibitor of NF-.kappa.B (SN) inhibited lipopolysaccharide (LPS)-induced nitric oxide production, a marker of DC maturation. Treatment of bone marrow-derived DC progenitors with NF-.kappa.B SN selectively suppressed the cell-surface expression of costimulatory mols. without interfering with MHC class I or class II expression. Furthermore, NF-.kappa.B SN DC induced allogeneic donor-specific hyporesponsiveness in mixed lymphocyte cultures, and this was assocd. with inhibition of Th1-type cytokine production. Finally, infusion of NF-.kappa.B SN-matured bone marrow-derived DC into allogeneic recipients prior to heart transplantation resulted in significant prolongation of allograft survival in the absence of immunosuppression. Specific interference with NF-.kappa.B and other transcriptional pathways involved in immune stimulation by DC using SN decoy approaches [illegible] promising alternative therapeutic modalities in organ transplantation [illegible].

REFERENCE COUNT: [illegible] THERE ARE [illegible] THIS REFERENCE AVAILABLE FOR THIS FORMAT. [illegible] AVAILABLE IN THE RE FORMAT

L25 ANSWER 1 [illegible]
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] graft survival by **dendritic cells** [illegible] **NF-kappa B** [illegible] decoys and adenoviral vectors [illegible] CTLA4-Ig.
AUTHOR: [illegible]
CORPORATE SOURCE: Department of Surgery and Thomas E. Starzl Transplantation Institute, University of Pittsburgh Medical Center, University of Pittsburgh, Pittsburgh, PA 15213, USA.
CONTRACT NUMBER: AI41011 (NIAID)
SOURCE: JOURNAL OF IMMUNOLOGY, [illegible]
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: [illegible]
ENTRY MONTH: [illegible]
ENTRY [illegible]

AB [illegible] **dendritic cells** (DCs) [illegible] genetically engineered using adenoviral (Ad) vectors to express immunosuppressive molecules that promote T cell unresponsiveness. The success of these DCs for therapy of allograft rejection has been limited in part by the potential of the adenovirus to promote DC maturation and the inherent ability of the DC to undergo maturation following in vivo administration. DC maturation occurs via NF-kappaB-dependent mechanisms, which [illegible] double-stranded "decoy" oligodeoxyribonucleotides (ODNs) containing binding sites for NF-kappaB. Herein, we describe the combined use of NF-kappaB ODNs and rAd vectors encoding CTLA4-Ig (Ad CTLA4-Ig) to generate stably immature murine myeloid DCs that secrete the potent costimulation blocking agent. These Ad CTLA4-Ig-transduced CBA DCs exhibit markedly impaired allostimulatory ability and promote apoptosis of activated T cells. Furthermore, administration of Ad CTLA4-Ig CBA-derived donor DCs (CD7BL10; [illegible] before transplant significantly prolongs MHC-mismatched [illegible]; [illegible] **tolerogenicity**, [illegible] T cells, and [illegible]. Use of NF-kappaB antisense oligonucleotides [illegible] potent costimulation blocking [illegible] therapy of allograft rejection or [illegible] systemic immunosuppression.

L25 ANSWER [illegible] MEDLINE DUPLICATE 2
ACCESSION NUMBER: [illegible] MEDLINE
DOCUMENT NUMBER: [illegible]
TITLE: Manipulation of **dendritic cells** [illegible] transplantation and [illegible] disease.
AUTHOR: Lu Lina; Thomson Angus W
CORPORATE SOURCE: Thomas E. Starzl Transplantation Institute, Department of Surgery, University of Pittsburgh Medical School, Pittsburgh, Pennsylvania [illegible]
CONTRACT NUMBER: [illegible]
SOURCE: [illegible]

Journal code: [illegible]. ISSN: [illegible]-1337.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: [illegible]
ENTRY DATE: Entered STN: [illegible]
Last Updated on STN: [illegible]
Entered Medline: [illegible]

AB **Dendritic cells** [illegible] immune system [illegible] uniquely [illegible] antigen-presenting cells that initiate and regulate immune responses. Recent studies have greatly improved our understanding of DC development, differentiation, activation, and function. DC exist as distinct subsets that differ in their lineage affiliation, surface molecule expression, and anatomical location. These factors seem to determine the T-cell stimulatory signals and type of T cell response-T helper 1, T helper 2, or T regulatory- induced by DC (1). Evidence has accumulated that DC play an important role in both central and peripheral tolerance via various mechanisms, including induction of T-cell anergy, immune deviation, T regulatory cell activity, and promotion of activated T-cell apoptosis. Although many of the details of the molecular basis of DC **tolerogenicity** have yet to be elucidated, emerging information suggests that costimulatory molecule deficiency, expression of death-inducing ligands (in particular Fas [CD95] ligand), microenvironmental factors (in particular anti-inflammatory/immunosuppressive cytokines), and inhibition of nuclear transcription regulatory proteins (e.g., nuclear factor-kappaB) can impact **tolerogenic** potential of DC [illegible]. Manipulation of DC by cytokines at their maturation and differentiation, or genetic engineering of these cells to express immunosuppressive molecules, offers potential for therapy of alloreactive responses and autoimmune diseases. In this brief overview, we outline [illegible] of these strategies in experimental models. Space [illegible] "tolerogenic" DC [illegible]

L25 ANSWER [illegible] MEDLINE DUPLICATE 3
ACCESSION NUMBER: [illegible] MEDLINE
DOCUMENT NUMBER: [illegible] PubMed ID: 10963964
TITLE: Prolongation of cardiac allograft survival using **dendritic cells** treated with NF-kB decoy oligodeoxyribonucleotides.
COMMENT: Erratum in: Mol Ther [illegible] Sep;[illegible]:[illegible]
Erratum in: Zhai H [illegible] Chen H]
AUTHOR: Giannoukakis N; Phillips B A; Finen S; Chen C; Feng L; Harnaha J; Li W; Thomson A W; Fung J J; Robbins P D; Lu L
CORPORATE SOURCE: Department of Molecular Genetics and Biochemistry, University of Pittsburgh, Pittsburgh, Pennsylvania [illegible], USA.
CONTRACT NUMBER: [illegible]
SOURCE: [illegible]
PUB. TYPE: [illegible]
LANGUAGE: [illegible]
FILE SEGMENT: [illegible]
ENTRY MONTH: [illegible]
ENTRY DATE: [illegible]

AB **Dendritic cells** [illegible]

tolerogenicity [illegible] NF-kB-activated [illegible] NF-kB translocation to the nucleus. [illegible], we found that double-stranded oligodeoxynucleotides containing binding sites for NF-kB (NF-kB ODN) are efficiently internalized by bone marrow-derived DC and specifically inhibit NF-kB-dependent transcription of a reporter gene. Moreover, exposure of DC to these oligonucleotide decoys inhibited lipopolysaccharide (LPS)-induced nitric oxide production, a marker of DC maturation. Treatment of bone marrow-derived DC progenitors with NF-kB ODN selectively suppressed the cell-surface expression of costimulatory molecules without interfering with MHC class I or class II expression. Furthermore, NF-kB ODN DC induced allogeneic donor-specific hyporesponsiveness in mixed leukocyte cultures, and this was associated with inhibition of Th1-type cytokine production. Finally, infusion of NF-kB ODN modified bone marrow-derived DC into allogeneic recipients prior to heart transplantation resulted in significant prolongation of allograft survival in the absence of immunosuppression. Specific interference with NF-kB and other transcriptional pathways involved in immune stimulation in DC using ODN decoys represents a novel [illegible] approach to induce tolerance in organ transplantation.

L25 ANSWER 4 [illegible] MEDLINE
ACCESSION NUMBER: [illegible] MEDLINE
DOCUMENT NUMBER: [illegible] PubMed ID: 11875462
TITLE: [illegible] **dendritic cells** by T(S) [illegible]: the critical role of inhibitory receptors ILT3 and ILT4.
COMMENT: Comment in: Nat Immunol. 2002 Mar;3(3):215-7
AUTHOR: [illegible] Chang C C; Ciubotariu R; Manavalan J S; Yuan J; Colovai A I; Piazza P; Lederman S; Colonna M; Cortesini R; [illegible]-Fabera R; [illegible]-Fiol N
CORPORATE SOURCE: Department of Pathology, Columbia University, New York, NY 10032, USA.
SOURCE: Nat Immunol, (2002 Mar) 3 (3) 237-43.
Journal code: 100941354. ISSN: 1529-2908.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: [illegible] Priority Journals
ENTRY MONTH: [illegible]
ENTRY DATE: [illegible]

AB [illegible] a family of [illegible] expressed by human myeloid and **dendritic cells**. [illegible] T-cell anergy and alloantigen-specific T suppressor [illegible] up-regulation of ILT3 and ILT4 on monocytes and **dendritic cells**, rendering these antigen-presenting cells (APCs) **tolerogenic**. **Tolerogenic** APCs show reduced expression of costimulatory molecules and induce antigen-specific unresponsiveness in CD4+ T helper cells. Studies of human renal transplant recipients who have mixed-chimerism patterns have circulating DC cells, which include myeloid and [illegible] ILT3 and ILT4 in donor APCs. These findings [illegible] a key [illegible] immune regulation.

L25 ANSWER 5 OF 6 [illegible]
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] **tolerogenic dendritic cells** [illegible] **tolerogenicity**

[illegible] and methods for making the same
INVENTOR(S): Morelli, Paul T.; Lu, Lina; Giannoukakis, Nick
PATENT ASSIGNEE(S): University of Pittsburgh of the Commonwealth System of Higher Education, USA
SOURCE: PCT Int. Appl., 64 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
[illegible]	A	[illegible]	[illegible]	[illegible]

[illegible]

AB The present invention relates to a **tolerogenic** mammalian **dendritic cells** (DCs) and methods for the prodn. of the **tolerogenic** DCs. In addn., the present invention provides a method for enhancing **tolerogenicity** in a host comprising administering the **tolerogenic** mammalian DCs of the present invention to the host. The **tolerogenic** DCs of the present invention comprise an isolated nucleic acid which can inhibit **NF-.kappa.B** [illegible] in the **tolerogenic** [illegible] of the present invention may further comprise a viral vector, which is capable of infecting a host, which does not affect the **tolerogenicity** of the **tolerogenic** DCs when present therein. Enhancing **tolerogenicity** in a host is useful for prolonging the graft survival and for treating inflammatory related diseases, such as autoimmune diseases.

L[illegible] ANSWER [illegible] ON STN
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] mRNA microarray profiles of **tolerogenic dendritic cells**
AUTHOR(S): Susin-Pena Cortesini, M.; Pianna, F.; He, F.; Ciubotariu, R.; LaMaccia, T.; Dalla-Favera, R.; Cortesini, R.
CORPORATE SOURCE: Department of Surgery, Columbia University, New York, NY, USA
SOURCE: [illegible]
PUBLISHER: [illegible]
DOCUMENT TYPE: [illegible]
LANGUAGE: [illegible]
AB Dendritic cells [illegible] **tolerogenic**, [illegible] **tolerogenic** [illegible] **dendritic cells** [illegible] and T suppressor [illegible] immature **dendritic cells** [illegible] T suppressor

cells differentiate into mature dendritic cells with a distinct phenotype. The maturation of the dendritic cell

REFERENCES:

L15 ANSWER 1 OF 2 [illegible]

ACCESSION NUMBER: [illegible]

DOCUMENT NUMBER: [illegible]

TITLE: [illegible]

AUTHOR(S): [illegible]

CORPORATE SOURCE: Department of Molecular Genetics and Biochemistry, University of Pittsburgh, Pittsburgh, PA, 15261, USA

SOURCE: Molecular Therapy (2000), 1(5, Pt. 1), 430-437
CODEN: MTOHCK; ISSN: 1525-0016

PUBLISHER: Academic Press

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Dendritic cells (DC) classically promote immune responses but can be manipulated to induce antigen-specific hyporesponsiveness in vitro. The expression of costimulatory mols. (CD40, CD86, CD80) at the DC cell surface correlates with their capacity to induce or suppress immune responses. Expression of these mols. is assocd. with NF-.kappa.B-dependent transcription of their genes. In **tolerogenicity** has been assocd. with impaired NF-.kappa.B-dependent transcription of costimulatory genes as well as NF-.kappa.B translocation to the nucleus. In this report, we demonstrate that a phosphorothioate-stabilized oligodeoxynucleotide duplex containing a consensus site for NF-.kappa.B (NF-.kappa.B ODN) can efficiently be incorporated by bone marrow-derived DC and specifically inhibit NF-.kappa.B-dependent transcription of a reporter gene. Moreover, exposure of DC to the oligonucleotide decoys inhibited lipopolysaccharide (LPS)-induced nitric oxide prodn., a marker of DC maturation. Treatment of bone marrow-derived DC progenitors with NF-.kappa.B ODN selectively suppressed the cell-surface expression of costimulatory mols. without interfering with MHC class I or class II expression. Furthermore, NF-.kappa.B ODN DC induced allogeneic donor-specific hyporesponsiveness in mixed leukocyte cultures, and this was assocd. with inhibition of Th1-type cytokine prodn. Finally, infusion of NF-.kappa.B ODN-modified bone marrow-derived DC into allogeneic recipients prior to heart transplantation resulted in significant prolongation of allograft survival in the absence of immunosuppression. Specific interference with NF-.kappa.B and other transcriptional pathways involved in immune stimulation in DC using ODN decoy approaches may be one means to promote tolerance in clinical transplantation. [illegible]

REFERENCE COUNT: [illegible] THERE ARE [illegible] RECORDS REFERENCING ASSIGNABLE FOR THIS ARTICLE. [illegible] ARE AVAILABLE IN THE RE FORMAT

L15 ANSWER [illegible]

ACCESSION NUMBER: [illegible]

DOCUMENT NUMBER: [illegible]

TITLE: Use of **tolerogenic** dendritic cells for inducing **tolerogenicity** in a host and materials for making the same

INVENTOR(S): Robbins, Paul D.; Li, Wei; Giannoukakis, Nick

PATENT ASSIGNEE(S): The University of Pittsburgh of the Commonwealth System of Higher Education, USA

SOURCE: PCT Int. Appl., [illegible] pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

[illegible]

WO [illegible]	A2	[illegible]	WO 2001-US13661	20010427	
WO 2001083715	A3	20020214			

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US [illegible] A1 [illegible] [illegible]-[illegible]

PRIORITY APPLN. INFO.: [illegible]

AB The present [illegible] **tolerogenic** mammalian [illegible] of the **tolerogenic** DCs. In addition, the present invention provides a method for enhancing **tolerogenicity** in a host comprising administering the **tolerogenic** mammalian DCs of the present invention to the host. The **tolerogenic** DCs of the present invention comprise an oligodeoxyribonucleotide (ODN) which has one or more NF-.kappa.B binding sites. The **tolerogenic** DCs of the present invention may further comprise a viral vector, and preferably an adenoviral vector, which does not affect the **tolerogenicity** of the **tolerogenic** DCs when present therein. Enhanced **tolerogenicity** in a host is useful for prolonging foreign graft survival and for treating inflammatory related diseases, such as autoimmune diseases.

L14 ANSWER [illegible] OF 16
ACCESSION NUMBER:
TITLE:
INVENTOR(S):
PATENT ASSIGNEE(S):
SOURCE:
DOCUMENT TYPE:
LANGUAGE:
FAMILY ACC. NUM. COUNT:
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6469144	B1	20021022	US 1997-982683	19971201
CA 2249206	AA	19971009	CA 1997-2249206	19970331
US 2002128116	A1	[illegible]	US [illegible]	[illegible]
US [illegible]	A1	[illegible]	US [illegible]	[illegible]

PRIORITY APPLN. INFO.:
US 1996-14499P P 19960402
US 1996-14500P P 19960402
US 1996-14501P P 19960402
US 1997-29093 A3 19970331
US 1996-34665 B1 19990430

AB The author discl[illegible] the cloning and sequence characterization for the tumor necrosis factor family member Apo-3 and Apo-2 ligand inhibitor (Apo-2LI), an extracellular fragment of Apo-3 generated by alternative splicing. In addn., the author discloses the apoptotic function of Apo-3, a partial characterization of its signaling pathway, and tissue specificity for its expression.

REFERENCE COUNT: 65 THERE ARE 65 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 2 OF 16 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2002:770147 CAPLUS
DOCUMENT NUMBER: 137:[illegible]
TITLE: Apo-3 protein, a new member of TNFR family inducing apoptosis and related ligand and [illegible]
INVENTOR(S): [illegible]
PATENT ASSIGNEE(S): [illegible]
SOURCE: [illegible]
DOCUMENT TYPE: [illegible]
LANGUAGE: [illegible]
FAMILY ACC. NUM. COUNT:
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US [illegible]	B1	[illegible]	US [illegible]	[illegible]
US 2002165121	A1	[illegible]	US [illegible]	[illegible]

PRIORITY APPLN. INFO.:
US 1996-14499P P 19960402
US [illegible] P [illegible]
US [illegible] B1 [illegible]

AB Novel polypeptides, designated Apo-3, which are [illegible] inducing apoptosis are provided. Apo-3 is a member of the TNFR family exhibiting sequence [illegible] with TNFR1, and it contains, in addition certain extracellular cysteine-rich [illegible] The cDNA for Apo-3 [illegible] and the [illegible] Apo-3 [illegible]

CPP32/yama. Apo-3 can activate NF-kappa-B activation.
REFERENCE COUNT: 2[illegible] THERE ARE 2[illegible] CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 3 OF 14 CAPLUS COPYRIGHT [illegible] ACS
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible]
INVENTOR(S): [illegible]
PATENT ASSIGNEE(S): [illegible]
SOURCE: [illegible]
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6414233	B1	20020903	US 1999-314691	19990519
PRIORITY APPLN. INFO.:			US 1998-85997P P	19980519
			US 1998-99066P P	19980903

OTHER SOURCE(S): MARPAT 137:110319
AB The invention provides novel saponin mixts. and compns. which are isolated from the species Acacia victoriae and methods for their use. The compns. may [illegible] against a variety [illegible].
REFERENCE COUNT: [illegible] THERE ARE [illegible] CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 4 OF 14 CAPLUS COPYRIGHT [illegible] ACS
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: Inhibition of NF-kappaB by triterpene compositions
INVENTOR(S): Patterson, Cam; Perkins, Nicholas
PATENT ASSIGNEE(S): Research Development Foundation, USA
SOURCE: PCT Int. Appl., [illegible] pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO [illegible]	[illegible]	[illegible]	[illegible]	[illegible]
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM
RW: AT, BE, CH, CY, DE, DK, EA, ES, FI, FR, GB, GH, GM, GR, IE, IT, KE, LS, LU, MC, MW, NL, OA, PT, SD, SE, SL, SZ, TZ, UG, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
PRIORITY APPLN. INFO.:			[illegible]	[illegible]

OTHER SOURCE(S):

AB The invention provides [illegible] inflammatory [illegible] NF-κB/Rel proteins. The inhibit [illegible] may include [illegible] barrier moiety that renders the molecule [illegible] membrane permeable. The barrier may include [illegible] peptide, polymer, lipid, and/or aliphatic moiety. The [illegible] may also contain additional chem. functionalities. Methods of using these [illegible] prevent and treat a wide range of inflammatory disorders, esp., pulmonary inflammatory conditions are described.

L14 ANSWER 5 OF 16 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2002:391866 CAPLUS
DOCUMENT NUMBER: 136:396944
TITLE: Preparation of T cell like expressing HIV Tat and NFκB and the use of the cell in treatment
INVENTOR(S): Miyake, Hiroshi; Ikawa, Yuji; Ono, Masaaki
PATENT ASSIGNEE(S): Takara Bio Inc., Japan
SOURCE: PCT Int. Appl., [illegible]
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT:
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002041914	A1	20020530	WO 2001-JP10018	20011116
W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM			
RW:	GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG			
AU 2002014307	A5	20020603	AU 2002-14307	20011116

PRIORITY APPLN. INFO.: [illegible]

AB A [illegible] is provided for [illegible] HIV [illegible] and/or NF-κB. [illegible] and/or NF-κB [illegible].

REFERENCE COUNT: [illegible] THERE ARE [illegible] RECORDS AVAILABLE FOR THIS [illegible] ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 6 OF 16 [illegible] COPYRIGHT [illegible] ACS
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] Alternate survival using [illegible] with NF-κB/Rel by [illegible]
AUTHOR(S): [illegible]
CORPORATE SOURCE: [illegible]
SOURCE: [illegible]
PUBLISHER: [illegible]
DOCUMENT TYPE: [illegible]
LANGUAGE: [illegible]

AB Dendritic cells (DC) classically prime to immune responses but can be manipulated to induce antigen-specific hyporesponsiveness in vitro. The expression of costimulatory mols. "CD4", "CD8", "CD11c" at the DC cell surface correlates with their capacity to induce or suppress immune responses. Expression of these mols. is associated with NF-.kappa.B-dependent transcription of their genes. DC tolerogenicity has been assocd. with impaired NF-.kappa.B-dependent transcription of costimulatory genes as well as NF-.kappa.B [illegible] the nucleus. In this [illegible] NF-.kappa.B [illegible] bone marrow-derived DC [illegible] NF-.kappa.B [illegible] bone marrow. Moreover, exposure [illegible] lipopolysaccharide (LPS)-induced [illegible] DC maturation. Treatment of bone marrow-derived DC progenitors with NF-.kappa.B ODN selectively suppressed the cell-surface expression of costimulatory mols. without interfering with MHC class I or class II expression. Furthermore, NF-.kappa.B ODN treatment [illegible] hyporesponsiveness in mixed leukocyte culture, and this was assocd. with inhibition of Th1-type cytokine prodn. Finally, infusion of NF-.kappa.B ODN-modified bone marrow-derived DC into allogeneic recipients prior to heart transplantation resulted in significant prolongation of allograft survival in the absence of immunosuppression. Specific interference with NF-.kappa.B and other transcriptional pathways involved in immune stimulation in DC using ODN decoy approaches could be one means to promote tolerance induction in organ transplantation. (c) 2008 Academic Press.

REFERENCE COUNT: 24 THERE ARE 24 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER [illegible]

ACCESSION NUMBER: [illegible]

DOCUMENT NUMBER: [illegible]

TITLE: [illegible] and methods for making the same

INVENTOR(S): Robbins, Paul D.; Su, Lina; Giannoukakis, Nick

PATENT ASSIGNEE(S): University of Pittsburgh of the Commonwealth System of Higher Education, USA

SOURCE: PCT Int. Appl., 64 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001083713	A2	[illegible]	WO [illegible]	[illegible]
WO 2001083713	A3	[illegible]		

DESIGNATED STATES: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW

PRIORITY APPLN. INFO.: [illegible]

AB The present invention [illegible] pharmaceutical [illegible] (ODN) and methods [illegible]. In addn., the present invention [illegible] comprising administering [illegible]

[illegible] present invention [illegible] NF-.kappa.B [illegible] the present invention may further [illegible], and preferably an adenoviral vector, which [illegible] DNs when present [illegible] in a host is useful for prolonging [illegible] survival and for treating inflammatory related diseases, such as autoimmune diseases.

L14 ANSWER 8 OF 16 CAPLUS COPYRIGHT [illegible] ACS

ACCESSION NUMBER: 2001:489042 CAPLUS

DOCUMENT NUMBER: 135:87194

TITLE: Inhibition of glycogen synthase kinase 3.beta. (GSK-3.beta.) for inhibiting NF-.kappa.B, and therapeutic use

INVENTOR(S): Hoeflich, Klaus; Luo, Juan; Woodgett, Jim

PATENT ASSIGNEE(S): The Ontario Cancer Institute, Can.

SOURCE: PCT Int. Appl., [illegible] pp.
CODEN: [illegible]

DOCUMENT TYPE: [illegible]

LANGUAGE: [illegible]

FAMILY ACC. NUM. COUNT: [illegible]

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 200104[illegible]	A[illegible]	[illegible]	WO 2000-CA1578	20001221
W: AT, CA, [illegible], US				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR				
EP 1244461	A2	20021002	EP 2000-986951	20001221
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY, TR				
US 2001053351	A1	20011220	US 2000-747552	20001221

PRIORITY APPLN. INFO.: US 1999-172064P P 1999122[illegible]
WO 2000-CA1578 W 20001221

AB The activity of NF-kB is modulated through the activity of [illegible] NF-kB activity. Inhibition or down-regulation of [illegible] results in decreased NF-kB activity. Inappropriate activation of NF-kB has been linked [illegible] inflammatory [illegible] strategies [illegible] therapeutic [illegible] the treatment or prevention of [illegible] killing of tumor cells [illegible] inhibition, when administered [illegible] ligands of TNF [illegible] function are also provided.

L14 ANSWER [illegible] CAPLUS COPYRIGHT [illegible] ACS

ACCESSION NUMBER: [illegible] CAPLUS

DOCUMENT NUMBER: [illegible]

TITLE: Regulation of HIV-1 transcription

AUTHOR(S): Roebuck, Kenneth A.; Saifuddin, Mohammed

CORPORATE SOURCE: Department of Immunology/Microbiology, Rush Presbyterian-St. Luke's Medical Center, Chicago, IL, [illegible], USA

SOURCE: [illegible] Expression [illegible]
CODEN: [illegible]

PUBLISHER: [illegible]

DOCUMENT TYPE: [illegible]

LANGUAGE: [illegible]

AB A [illegible]

learned about the transcriptional regulation of the HIV-1 genome in infected cells. It has been demonstrated that HIV-1 transcription depends on a varied and complex interaction of host cell transcription factors with the viral long terminal repeat (LTR) promoter. The regulatory elements within the LTR interact with constitutive and inducible transcription factors to direct the assembly of a stable transcription complex that stimulates multiple rounds of transcription by RNA polymerase II (RNAPII). However, the majority of these transcripts terminate prematurely in the absence of the virally encoded transactivator protein Tat, which stimulates HIV-1 transcription elongation by interacting with a stem-loop RNA element (TAR) formed at the extreme 5' end of all viral transcripts. The Tat-TAR interaction recruits a cellular kinase into the initiation-elongation complex that alters the elongation properties of RNAPII during its transit through TAR. This review summarizes our current knowledge and understanding of the regulation of HIV-1 transcription in infected cells and highlights the important contributions human lentivirus gene regulation has made to our general understanding of the transcription process.

REFERENCE COUNT: [illegible] THERE ARE [illegible] CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 10 OF 16 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1999:657226 CAPLUS

DOCUMENT NUMBER: 132:161869

TITLE: Long terminal repeat promoter/enhancer activity of different subtypes of HIV type 1

AUTHOR(S): Naghavi, Mojgan H.; Schwartz, Stefan; Sonnerborg, Anders; Vahlne, Anders

CORPORATE SOURCE: Division of Clinical Virology, Department of Immunology, Microbiology, Pathology, and Infectious Diseases, Karolinska Institute, Huddinge University Hospital, Stockholm, S-141 86, Swed.

SOURCE: AIDS Research and Human Retroviruses (1999), 15(14), [illegible]

CODEN: ARHRE7; ISSN: 0889-2229

PUBLISHER: Mary Ann Liebert, Inc.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Transcription of the HIV-1 provirus genome is regulated by a complex interplay between viral regulatory proteins and cellular transcription factors that interact with the viral long terminal repeat (LTR) region of HIV-1. However, several cellular transcription factors have been identified that can interact with the HIV-1 LTR; the significance of all of these factors is not clearly understood. In this study the authors have characterized the LTR region of different subtypes of HIV-1 with regard to nucleotide sequence and promoter activity. The LTR regions of HIV-1 from peripheral [illegible] originating from [illegible] different [illegible] were sequenced and further [illegible] subtype-specific LTR [illegible] subtype [illegible] subtype-specific sequences. Interestingly, [illegible] subtype [illegible] LTRs [illegible] subtypes [illegible] A, B, C, D, and [illegible] NF-kappa.B sites [illegible] subtype C LTR may be less potent. Thus, [illegible] genetic diversity of the LTR may result in HIV-1 subtypes with different replicative properties.

REFERENCE COUNT: [illegible] THERE ARE [illegible] CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 AN [illegible]
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: NF-.kappa.B [illegible] crosslinking to DNA duplexes, [illegible] pyrophosphate [illegible]
AUTHOR(S): Kuprash, Dmitry A.; Pastorova, Olga A.; Gottikh, Marina B.; Tukalo, Mikhail; Mainy, Claude; Shakarova, Zoe A.
CORPORATE SOURCE: A.N. Belozersky Institute of Physical and Chemical Biology and Department of Chemistry, Moscow State University, Moscow, 119899, Russia
SOURCE: FEBS Letters (1996), 381(1,2), 35-8
CODEN: FEBLAL; ISSN: 0014-5793
PUBLISHER: Elsevier
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A new express technique based on the use of [illegible] synthesizing DNA duplexes contg. non-substituted or phosphorothioate pyrophosphate internucleotidic bridges is described. This technique was used to prep. DNA duplexes bearing a photoreactive dA residue at different positions in the human NF-.kappa.B binding site and a complementary sequence in HIV-1 5'-[illegible]-3'. These duplexes were found to bind within the ternary complexes formed by the homodimer of NF-.kappa.B p50 subunit complexed with the corresponding oligonucleotides. The crosslinking of NF-.kappa.B p50 subunit to the DNA duplexes only, a monosubstituted pyrophosphate internucleotidic bridge was successfully performed.

L14 ANSWER [illegible] COPYRIGHT [illegible] ACS
ACCESSION NUMBER: 1994:696026 CAPLUS
DOCUMENT NUMBER: 121:296026
TITLE: Determination of the binding of transcription factors to nucleic acids by immunoassay
INVENTOR(S): Doppler, Clemens; Hilpert, Matthias; Stockinger, Hubertus
PATENT ASSIGNEE(S): Boehringer Mannheim G.m.b.H., Germany
SOURCE: Eur. Pat. Appl., 9 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
R: [illegible]				
DE [illegible]	A1	[illegible]	DE [illegible]	[illegible]
PRIORITY APPLN. INFO.:			DE [illegible]	19930416

AB Binding of a transcription factor to a nucleic acid is detd. by (a) immobilizing either the nucleic acid or the transcription factor on a solid phase; (b) incubating the transcription factor with the nucleic acid; (c) adding a labeled antibody against either the nucleic acid or the transcription factor under conditions allowing binding; (d) sepg. the solid and liq. phases; and (e) measuring the amt. of label in either phase. Thus, binding of transcription factor NFKB in nuclear extr. of PMA-PHA-activated Jurkat cells to a synthetic oligonucleotide contg. a binding site for NFKB was detd. by incubating the oligonucleotide biotinylated at the 5'-end, immobilizing the complex formed between NFKB and the biotinylated oligonucleotide on streptavidin-coated microtiter plates, adding a digoxigenin-labeled monoclonal antibody against NFKB, and measuring the amt. of digoxigenin-labeled antibody bound to the solid phase.

L14 AN [illegible]

ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: In vitro study of functional interaction of Sp1, NF-.kappa.B/Rel, and AP1 in phorbol 12-myristate 13-acetate-responsive human immunodeficiency virus type 1 long terminal repeat activation.
AUTHOR(S): Li, Yuzhi; Kao, Chris; Brasier, Allan R., Jr.
CORPORATE SOURCE: Cold Spring Harbor Lab., Cold Spring Harbor, New York, NY, 11724, USA
SOURCE: [illegible]
PUBLISHER: [illegible]
DOCUMENT TYPE: [illegible]
LANGUAGE: English
AB We examd. the cooperative activity between the Sp1 and NF-.kappa.B/Rel sites of the human immunodeficiency virus type 1 long terminal repeat in response to phorbol 12-myristate 13-acetate (PMA) stimulation in an in vitro transcription assay. Sp1 sites alone do not account for the activation induced by PMA. When mutations in Sp1 sites were combined with mutations in the NF-.kappa.B/Rel sites, a dramatic redn. in PMA-induced transcriptional activity was obsd. This redn. was much greater than the redn. assocd. with mutations involving only the NF-.kappa.B/Rel sites. This finding suggests that there is functional cooperation between Sp1 and NF-.kappa.B/Rel and that this is one possible mechanism for transcription activation by NF-.kappa.B/Rel. The three AP1 sites in the U3 regulatory region of the long terminal repeat, however, seem to be uninvolved in the earliest moments of transcriptional activation by PMA.

L14 ANSWER 11 OF [illegible] MEDLINE on STN
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: Transcriptional activation of human immunodeficiency virus type 1 gene expression.
AUTHOR(S): Berkhout, Ben; Arrigo, Steven J.; Darlix, Jean-Luc; Gatignol, Anne; Silver, Jerry
CORPORATE SOURCE: Howard Hughes Medical Institute, University Michigan, Ann Arbor, MI, 48109-0620, USA
SOURCE: Journal of Virology (1994), 68(10), 6620-3
CODEN: JOVIAM; ISSN: 0022-538X
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Human immunodeficiency virus type 1 (HIV-1) gene expression is regulated by an enhancer region composed of multiple potential cis-acting regulatory sites. Here, we describe binding sites for the transcription factor Sp1 in the HIV-1 long terminal repeat which mediate Tat transactivation. One site is embedded within the two previously described kappa B elements, and a second site is located further upstream. Mutations in footprinting and in vitro transcription assays [illegible]

L14 ANSWER 12 OF [illegible] MEDLINE on STN
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible]
AUTHOR(S): [illegible]

CORPORATE SOURCE: [illegible]
SOURCE: [illegible]
DOCUMENT TYPE: [illegible]
LANGUAGE: [illegible]
AB In particular, DNA [illegible] DNA-binding proteins (AT-BP1 and AT-BP2) were [illegible]. These proteins, which preferentially bind to lambda.gt11 lysogens, bind to the B-domain of the .alpha.1-antitrypsin promoter, an element which is important for the liver-specific expression of .alpha.1-antitrypsin. Anal. of the cDNA sequences encoding these proteins reveals that both contain 2 zinc fingers of the Cys2-His2 type followed by a highly acidic stretch of 20 amino acids. AT-BP1 contains a 2nd putative DNA-binding domain consisting of an 8-fold repeat of a SPKK (Ser-Pro-Lys/Arg-Lys/Arg) motif. Both proteins bind to the NF.kappa.B recognition site in the MHC gene enhancer with significant higher affinity than to the .kappa. immunoglobulin gene enhancer, or to the B-domain of the .alpha.1-antitrypsin gene promoter. Anal. of mRNA expression shows that AT-BP1 and AT-BP2 are expressed in all the tissues examd. While the physiol. roles of AT-BP1 and AT-BP2 remain to be elucidated, their predicted amino acid sequences [illegible] DNA-binding motifs suggest that they may be involved in [illegible].

L14 AN [illegible]
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: UV-induced DNA damage is an intermediate step in UV-induced expression of human immunodeficiency virus type 1, collagenase, c-fos, and metallothionein
AUTHOR(S): Stein, Bernd; Rahmsdorf, Hans Jobst; Steffen, Anja; Litfin, Margarethe; Herrlich, Peter
CORPORATE SOURCE: Inst. Genet. Toxikol., Kernforsch. Karlsruhe, Karlsruhe, D-7500/1, Fed. Rep. Ger.
SOURCE: Molecular and Cellular Biology (1989), 9(11), 5169-81
CODEN: MCEBD4; ISSN: 0270-7306
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The primary target of relevant UV absorption, the pathways leading to gene activation, and the elements mediating the UV-inducible signal in the human immunodeficiency virus type 1 (HIV-1) long terminal repeat, in the gene coding for collagenase, and in the human c-fos gene were [illegible]. [illegible] UV-induced DNA damage is necessary and sufficient to mediate UV induction of these genes. Furthermore, the UV-inducible activity of the HIV-1 enhancer is dependent on sequences located between nucleotide positions -104 and -79 of HIV-1, between positions -72 and -61 of the collagenase gene, and between positions -320 and -299 of fos). These sequences have no apparent homology to each other and bind different trans-acting factors; a member of the NF.kappa.B family binds to the HIV-1 enhancer, the heterodimer of Jun and Fos (AP-1) binds to the collagenase enhancer, and the ubiquitous factors pEF and pEF bind to fos. DNA-binding activities of the factors recognizing the HIV-1 and collagenase enhancers are augmented in extracts from UV-treated cells. The increase in activity is due to post-translational modification. Whereas AP-1 resides in the nucleus and must be translocated there, NF.kappa.B is activated in the cytoplasm, indicating the existence of a cytoplasmic signal transduction pathway triggered by UV-induced DNA damage. In addition to activation, new synthesis of AP-1 is induced by UV irradiation.

PALM INTRANET

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L6 ANSWER 1 OF 9 BIOSIS COPYRIGHT [illegible] BIOLOGICAL ABSTRACTS INC.[illegible]

ACCESSION NUMBER: 2002:345247 BIOSIS

DOCUMENT NUMBER: PREV200200345247

TITLE: Allochimeric class I MHC protein-induced tolerance by partial TCR engagement [illegible]

AUTHOR(S): [illegible]

CORPORATE SOURCE: [illegible] University of Texas at Houston, Houston, TX, [illegible] stepkowski@uth.tmc.edu USA

SOURCE: Transplantation (Baltimore), (April 27, 2002) Vol. 73, No. [illegible], pp. [illegible]. http://www.transplantjournal.com/. print. ISSN: 0041-1337.

DOCUMENT TYPE: Article

LANGUAGE: English

AB Background: The [illegible] toxicities associated with the general immune suppression resulting from current clinical immunosuppressive therapies continue to plague transplant recipients as well as jeopardize allograft survival. Methods: The present study utilized allochimeric class I MHC antigens (alpha1h u70-77-RT1.Aa) bearing only four donor RT1.Au polymorphic amino acids (a.a.; His70, Val73, Asn74, and Asn77) superimposed on the recipient RT1.Aa background to induce transplantation tolerance in the rat cardiac transplant model. Results: Oral delivery of alpha1h u70-77-RT1.Aa protein alone (days [illegible]) induced tolerance, as evidenced by inhibition of both acute and chronic rejection processes. Delivery of alpha1h u70-77-RT1.Aa with [illegible] doses of cyclosporine (CsA) also prevented chronic rejection, otherwise readily developed after treatment with CsA [illegible]. A [illegible]-based analysis [illegible] interleukin [illegible] cells and elevated numbers [illegible]. Adoptive transfer experiments revealed that [illegible] T cells mediated tolerance. The same T cells displayed diminished T cell receptor (TCR)-driven signaling via extracellular regulated kinase, AP-1, and NF-kappaB, as well as the common gamma-chain (gammac) cytokine-receptor-induced signaling by Janus kinase 3 (Jak3)/stimulators and activators of transcription Stat/5 pathways. Tolerance induction was prevented in vivo by inhibition of signal 2 by CTLA4Ig or of signal 3 by either rapamycin, which disrupts the mammalian target of rapamycin, or AG490, which inhibits Jak3. Finally, partial or complete tyrosine phosphorylation of [illegible] was observed in alloantigen-specific T cell clones in response to **tolerogenic** versus immunogenic peptides, respectively. Conclusions: Tolerance induction by allochimeric proteins is achieved by partial TCR [illegible] in the presence of signals 2 and 3, resulting in a skewed [illegible] phenotype.

L6 ANSWER 2 OF 9 BIOSIS COPYRIGHT [illegible] BIOLOGICAL ABSTRACTS INC.[illegible]

ACCESSION NUMBER: [illegible] BIOSIS

DOCUMENT NUMBER: [illegible]

TITLE: [illegible]

AUTHOR(S): [illegible]

CORPORATE SOURCE: [illegible] Transplantation Institute, University of Pittsburgh [illegible] Lothrop Street, E1[illegible], [illegible] Tower, Pittsburgh, PA, [illegible] USA

SOURCE: Transplantation (Baltimore), (January [illegible]) Vol. [illegible], No. [illegible], pp. [illegible]. http://www.transplantjournal.com/. print. ISSN: 0041-1337.

DOCUMENT TYPE: General Review

LANGUAGE: English

AB Dendritic cells (DC) constitute a [illegible] antigen-presenting cells [illegible]

[illegible] DC [illegible] exist as distinct subsets that differ in their lineage affiliation, surface molecule expression, and T cell activation. These factors seem to determine the T-cell polarizing signals and type of T cell response-T helper 1, T helper 2, or T regulatory cell-induced by DC (1). Evidence has accumulated that DC play an important role in both central and peripheral tolerance via various mechanisms, including induction of T-cell anergy, immune deviation, T regulatory cell activity, and promotion of activated T-cell apoptosis. Although many of the details of the molecular basis of DC **tolerogenicity** have yet to be elucidated, emerging information suggests that costimulatory molecule deficiency, expression of death-inducing ligands (in particular Fas (CD95) ligand), microenvironmental factors (in particular anti-inflammatory/immunosuppressive cytokines), and inhibition of NF-κB transcription regulatory proteins, e.g., by natural [illegible] can impart **tolerogenic** properties to DC. Manipulation of DC by one or more of these factors, or other as yet undefined mechanisms, offers potential for therapy of allograft rejection and autoimmune disease. In this brief overview, we outline principles and methods for generation of **"tolerogenic"** DC and outcomes that have been reported in experimental models. Space constraints limit literature citations.

L6 ANSWER 3 OF [illegible] BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 3
ACCESSION NUMBER: [illegible] BIOSIS
DOCUMENT NUMBER: PREV[illegible]
TITLE: Reduced expression of NF-AT and NF-kappaB transcription factors in tolerant recipients treated with **tolerogenic** allochimeric donor/recipient class I MHC protein.
AUTHOR(S): Akioka, K. (1); Kirkin, A.; Wang, M.; Tian, L.; Yu, J.; Stepkowski, S. M.; Nahan, B. D.
CORPORATE SOURCE: (1) Division of Immunology and Organ Transplantation, University of Texas Medical School at Houston, [illegible] Pressler St., Houston, TX, [illegible]
SOURCE: Transplantation [illegible], [illegible], [illegible], [illegible], [illegible].
[illegible]
DOCUMENT TYPE: [illegible]
LANGUAGE: [illegible]
SUMMARY LANGUAGE: [illegible]

L6 ANSWER 4 OF [illegible] BIOSIS COPYRIGHT 2[illegible] BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: [illegible] BIOSIS
DOCUMENT NUMBER: PREV[illegible]
TITLE: Marked prolongation of cardiac allograft survival by dendritic cells retrovirally engineered with NF-kappaB oligodecoyrix while they are in vitro and with viral vectors encoding CTLA4-Ig.
AUTHOR(S): Benham, A. M.[illegible]; Isobe, Tadashi; Ikeda, Hiroyuki; Tsai, Zongyou; Wang, Lianli; Xu, Yibing; Hancock, W. [illegible]; Fairchild, Paul J.; Tone, Mitsuo; Tone, Yuko; [illegible], [illegible]; Lu, Lina
[illegible]
[illegible]
[illegible]

DOCUMENT TYPE: Article
LANGUAGE: English
AB Bone marrow-derived dendritic cells (DCs) can be genetically engineered using adenoviral (Ad) vectors to express immunosuppressive molecules that promote T cell unresponsiveness. The success of DC therapy in allograft rejection has been limited in part by the inefficiency of the adenovirus 5 gene transfer to DCs and the transient ability of the DC to [illegible] Here we report the generation of [illegible] DCs by [illegible] blocking agent, [illegible]. Both, by themselves, [illegible] NF-kappaB [illegible] to [illegible] CTLA4-Ig (Ad CTLA4-Ig) to generate stably immature myeloid DCs that secrete the potent costimulation blocking agent. These Ad CTLA4-Ig-transduced ODN DCs exhibit markedly impaired allostimulatory ability and promote apoptosis of activated T cells. Furthermore, administration of Ad CTLA4-Ig ODN-treated donor DCs (C57BL/10; B10/H-2k) before transplant significantly prolongs MHC-mismatched (BALB/c; BALB/H-2K) vascularized heart allograft survival, with long-term (>100 days) donor-specific graft survival in 40% of recipients. The mechanism(s) responsible for DC **tolerogenicity**, which may involve activation-induced apoptosis of alloreactive T cells, do not lead to skewing of intragraft Th cytokine responses. Use of NF-kappaB antisense decoys in conjunction with rAd encoding a potent costimulation blocking agent offers promise for therapy of allograft rejection or autoimmune disease with minimization of systemic immunosuppression.

L6 ANSWER [illegible] OF [illegible] CAPLUS COPYRIGHT [illegible] ACS
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] **tolerogenic** [illegible]
[illegible] **tolerogenicity** [illegible]
[illegible]
INVENTOR(S): [illegible]; Fu, Yang; Thannickal, Nick
PATENT ASSIGNEE(S): University of Pittsburgh of the Commonwealth System of Higher Education, USA
SOURCE: PCT Int. Appl., 64 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001083718	A2	20011108	[illegible]	[illegible]
WO 2001083718	A3	20020314		

W: [illegible]
RW: [illegible]
[illegible]
PRIORITY APPLN. INFO.: [illegible]
AB The present invention relates to **tolerogenic** mammalian dendritic cells (DCs) and methods of preparing the **tolerogenic** DCs. In addition, the present invention provides a method for enhancing **tolerogenicity** in a subject comprising administering the **tolerogenic** mammalian DCs of the present invention to the subject. The **tolerogenic** DCs of the present invention comprise [illegible]

NF-.kappa.B [illegible] **tolerogenic** [illegible] the present invention may further comprise a third [illegible], and preferably an additional [illegible], which [illegible] the **tolerogenicity** of the **tolerogenic** [illegible] such grafts. Enhanced **tolerogenicity** [illegible] useful for prolonging foreign graft survival and [illegible] inflammatory related diseases, such as autoimmune diseases.

L6 ANSWER 6 OF 9 CAPLUS COPYRIGHT 2[illegible] ACS
ACCESSION NUMBER: 2001:922841 CAPLUS
DOCUMENT NUMBER: 137:45987
TITLE: The role of Stat5 in the induction of [illegible] T cells in transplantation tolerance
AUTHOR(S): Stepkowski, S. M.; Kirken, R. A.; Tary, G. [illegible]; Trawick, F. K.; Wang, M.; Dilger, N.; Wang, M.-F.; Tian, L.; Chang, T.; Zhang, B. L.
CORPORATE SOURCE: [illegible] Department of Internal Medicine, University of Texas, Houston, TX, USA
SOURCE: [illegible] (2), [illegible], 3[illegible]-3[illegible] CODEN: [illegible]; ISSN: [illegible]
PUBLISHER: [illegible]
DOCUMENT TYPE: [illegible]
LANGUAGE: English
AB The roles of extracellular signal-regulated kinase 2 (Erk2), NF-.kappa.B, AP-1, Janus tyrosine kinase 3 (Jak3), and stimulators and activators of transcription 5 (Stat5) in mediating transplantation tolerance were studied. Tolerant recipients that had carried functional Wistar-Furth (WF) grafts for more than 100 days were rechallenged with a second WF heart. Purified T cells from spleens and lymph nodes of rejectors and tolerant T cells showed a significant increase in Jak3 expression. The presence of phosphorylated Erk2 and the expression of Jak3 indicated that the tolerant animals have actively reacting alloantigen-specific T cells, but that their response was distinct from that in nonactivated T cells of syngeneic grafts or fully activated T cells in rejectors. Tolerant T cells showed almost undetectable AP-1, NF-.kappa.B, and Stat5 DNA binding activities. The IL-2 plus stimulation with immunoadhesin B7-2 [illegible] showed potent Stat5 DNA binding [illegible] by IL-2 [illegible] by IL-1. On the other hand, the [illegible] with **tolerogenic** B7-2 [illegible] [illegible] Stat5 DNA binding [illegible] IL-2 stimulation. These results indicate the [illegible] IL-2 [illegible] [illegible] with partial TCR activation.
REFERENCE COUNT: [illegible] THERE ARE [illegible] CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER [illegible] CAPLUS COPYRIGHT [illegible] ACS
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] mRNA microarray profiles of **tolerogenic** [illegible] cells
AUTHOR(S): Turka-Feda Cortesini, M.; Bianca, F.; [illegible], E.; Ciabotariu, R.; LaMattina, J.; Della-Pietra, F.; Cortesini, R.
CORPORATE SOURCE: Department of [illegible], [illegible] University, New York, NY, USA
SOURCE: [illegible] Immunol. [illegible], [illegible], [illegible] [illegible]; CODEN: [illegible]
PUBLISHER: [illegible]
DOCUMENT TYPE: [illegible]
LANGUAGE: [illegible]
AB [illegible] **tolerogenic**, [illegible]

changes occurring in **tolerogenic** APC, the mRNA profile of NF-[illegible] dendritic cells exposed to allospecific T helper and T suppressor [illegible] were analyzed. This study now provides evidence that immature dendritic cells stimulated by T suppressor cells differentiate into mature dendritic cells with a distinct phenotype. The identification of the signaling pathways of dendritic cell differentiation will [illegible] the development of new therapeutic strategies.

REFERENCE FORMATS: [illegible] AVAILABLE FOR THIS [illegible] AVAILABLE IN THE 96 FORMAT

L6 ANSWER [illegible] [illegible] [illegible] [illegible]
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: Prolongation of cardiac allograft survival using dendritic cells treated with NF-.kappa.B decoy oligodeoxyribonucleotides
AUTHOR(S): Hamaguchi, Nina; Donham, C. Andrew; Qian, Shiguang; Zhou, Zhongyou; Peng, Lansha; Harnaha, Jo; Li, Wei; Thomson, Angus W.; Fung, John J.; Robbins, Paul D.; Lu, Lina
CORPORATE SOURCE: Department of Molecular Genetics and Biochemistry, University of Pittsburgh, Pittsburgh, PA, 15261, USA
SOURCE: Molecular Therapy (2003), 1,5, Pt. 1, 4[illegible]-43[illegible]
CODEN: MTOHCK; ISSN: 1[illegible]-0[illegible]
PUBLISHER: Academic Press
DOCUMENT TYPE: Journal

L5 ANSWER [illegible] ABSTRACTS INC.DUPLICATE [illegible]
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] **dendritic cells** [illegible] tolerance [illegible] in transplantation and autoimmune disease.
AUTHOR(S): [illegible]; [illegible], Angus W.
CORPORATE SOURCE: [illegible] Thomas E. Starzl Transplantation Institute, University of Pittsburgh Medical Center, [illegible] Lothrop Street, E1554, Biomedical Science Tower, Pittsburgh, PA, [illegible]: Lul@msx.upmc.edu USA
SOURCE: Transplantation (Baltimore), (January [illegible], 2002) Vol. [illegible], No. 1 Supplement, pp. [illegible]. http://www.transplant[illegible]ournal.com/. print. ISSN: [illegible].
DOCUMENT TYPE: General Review
LANGUAGE: [illegible]

AB **Dendritic cells** [illegible] un[illegible] that initiate and regulate immune [illegible] have improved our understanding of DC [illegible], differentiation, [illegible], and function. DC exist as distinct subsets that differ in their lineage affiliation, surface molecule expression, and biological function. These factors seem to determine the T-cell polarizing signals and type of T cell response-T helper 1, T helper [illegible], or T regulatory-induced by DC (1). Evidence has accumulated that DC play an important role in both central and peripheral tolerance via various mechanisms, including induction of T-cell anergy, immune deviation, T regulatory cell activity, and promotion of activated T-cell apoptosis. Although many of the details of the molecular basis of DC **tolerogenicity** have yet to be elucidated, emerging information suggests that costimulatory molecule deficiency, expression of death-inducing ligands (in particular Fas (CD95) ligand), microenvironmental factors (in particular anti-inflammatory/immunosuppressive cytokines), and inhibition of gene transcription regulatory proteins (e.g., nuclear factor-kappaB) can impart **tolerogenic** potential [illegible] DC (2). Manipulation of DC by control of their maturation and differentiation, [illegible] cells to express immunosuppressive [illegible], [illegible] potential for therapy of [illegible] graft [illegible] and [illegible] disease. In this brief [illegible], we outline [illegible] **"tolerogenic"** DC and [illegible] experimental models. [illegible]

L5 ANSWER [illegible] ABSTRACTS INC.
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] of cardiac allograft survival by **dendritic cells** [illegible] engineered with NF-kappaB oligodeoxyribonucleotide decoys and [illegible] TRAIL-Ig.
AUTHOR(S): Bonham, C. Andrew; [illegible]; [illegible]; Chen, [illegible]; Wang, [illegible]; Ma, [illegible]; [illegible]; Robbins, Paul D.; Thomson, Angus W.; [illegible]; [illegible]; Lu, [illegible]
CORPORATE SOURCE: [illegible] Thomas E. Starzl Transplantation Institute, University of Pittsburgh Medical Center, [illegible], Biomedical Science Tower, [illegible], Pittsburgh, PA, [illegible]
SOURCE: [illegible]
DOCUMENT TYPE: [illegible]
LANGUAGE: [illegible]
AB [illegible] **dendritic cells** [illegible]

genetically [illegible] using [illegible] immunosuppressive [illegible]. The success [illegible] has been limited in part [illegible] maturation and [illegible] in vivo administration [illegible] NF-kappa B-dependent mechanisms, [illegible] "decoy" [illegible] oligodeoxyribonucleotides (ODN) [illegible] NF-kappa B. Herein, we describe the [illegible] NF-kappa B ODN and rAd vectors encoding CTLA4-Ig (Ad CTLA4-Ig) [illegible] immature murine myeloid DCs that secrete the potent costimulation blocking agent. These Ad CTLA4-Ig-transduced ODN DCs exhibit markedly impaired allostimulatory ability and promote apoptosis of activated T cells. Furthermore, administration of Ad CTLA4-Ig ODN-treated donor DCs (C57BL10; B10(H-2b)) before transplant significantly prolongs MHC-mismatched (C3HHeJ; C3H(H-2k)) vascularized heart allograft survival, with long-term (>100 days) donor-specific graft survival in [illegible] recipients. The mechanism(s) responsible for DC **tolerogenicity**, which may involve activation-induced apoptosis of allo-reactive T cells, [illegible] not lead to skewing of intragraft Th cytokine responses. [illegible] NF-kappa B antisense decoys in conjunction with rAd encoding a potent costimulation blocking agent offers promise for therapy of allograft rejection [illegible] autoimmune disease with minimization of systemic immunosuppression.

L8 ANSWER [illegible]
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] **tolerogenic dendritic cells** for enhancing **tolerogenicity** in a host and methods for making the same
INVENTOR(S): Robbins, Paul D.; Lu, Lina; Giannoukakis, Nick
PATENT ASSIGNEE(S): University of Pittsburgh of the Commonwealth System of Higher Education, USA
SOURCE: PCT Int. Appl., [illegible] pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO [illegible]	A[illegible]	[illegible]	[illegible]	[illegible]
WO [illegible]	A[illegible]	[illegible]		

W: [illegible] TM
RW: [illegible] SE, TR, BF, [illegible] TG
[illegible]

PRIORITY APPLN. INFO.: [illegible]

AB The present invention relates to [illegible] **tolerogenic** mammalian **dendritic cells** (DCs) and methods for the production of the **tolerogenic** DCs. In addition, the present invention provides a method for enhancing **tolerogenicity** in a host comprising administering the **tolerogenic** mammalian DCs of the present invention to the host. The **tolerogenic** DCs of the present invention comprise an [illegible] NF-.kappa.B binding [illegible]. The **tolerogenic** DCs of the present invention may further [illegible] **tolerogenicity** [illegible]

the **tolerogenic** DCs when presented [illegible]. Enhanced **tolerogenicity** in a host is useful [illegible] survival and for treating inflammatory [illegible], such as autoimmune diseases.

L2 ANSWER 4 OF [illegible]
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible]
tolerogenic dendritic cells
AUTHOR(S): [illegible]
CORPORATE SOURCE: Department of Pathology, Columbia University, New York, NY, USA
SOURCE: Human Immunology (2001), 62(10), 1065-1072
CODEN: HUIMDV; ISSN: 0198-8859
PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB **Dendritic cells** are crucial to the activation as well as suppression of the immune response. Previous reports have illustrated that APC interacting with antigen-specific T suppressor cells become **tolerogenic**, inducing T helper anergy. To characterize the molecular changes occurring in **tolerogenic** APC, the mRNA profile of MHC-I **dendritic cells** exposed to allospecific T helper and T suppressor cells was analyzed. This study [illegible] that immature **dendritic cells** [illegible] suppressor cells [illegible] **dendritic cells** with a distinct gene [illegible] the signaling pathways of **dendritic cell** [illegible] the development of [illegible] therapeutic strategies.
REFERENCE COUNT: 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 5 OF [illegible] COPYRIGHT 2004 ACS
ACCESSION NUMBER: [illegible]:193192 CHEMCS
DOCUMENT NUMBER: 135:41141
TITLE: Prolongation of cardiac allograft survival using **dendritic cells** treated with NF-.kappa.B decoy oligodeoxyribonucleotides
AUTHOR(S): Giannoukakis, Nick; Bonham, C. Andrew; Qian, Shiguang; Zhou, Zhongyou; Penn, Lansha; Harnaha, J.; Li, Wei; Thomson, Angus W.; Rudy, Bela K.; Robbins, Paul D.; Lu, Libo
CORPORATE SOURCE: [illegible]
SOURCE: [illegible]
PUBLISHER: [illegible]
DOCUMENT TYPE: [illegible]
LANGUAGE: [illegible]
AB **Dendritic cells** [illegible] **tolerogenicity** [illegible] dependent transcription [illegible] translocation to the nucleus. In this report, we [illegible] double-stranded [illegible] NF-.kappa.B (NF-.kappa.B ODN) [illegible] marrow-derived [illegible] NF-.kappa.B-[illegible]

transcription [illegible]. However, exposure of DC to the [illegible] lipopolysaccharide (LPS)-induced nitric oxide [illegible]. Treatment of bone marrow-derived DC precursors with NF-.kappa.B [illegible] suppressed the [illegible] expression of costimulatory molecules without interfering with MHC class I or class II expression. Furthermore, NF-.kappa.B ODN DC induced allogeneic non-specific hyporesponsiveness in mixed leukocyte cultures, and this was assocd. with inhibition of Th1-type cytokine prodn. Finally, infusion of NF-.kappa.B ODN-modified bone marrow-derived DC into allogeneic recipients prior to heart transplantation resulted in significant prolongation of allograft survival in the absence of immunosuppression. Specific interference with NF-.kappa.B and other transcriptional pathways involved in immune activation by DC might [illegible] decoy approaches could be [illegible] prevention and treatment of organ transplant [illegible]. (C) 2001 Academic Press.

REFERENCE COUNT: [illegible] THERE ARE [illegible] OTHER REFERENCES AVAILABLE FOR THIS RECORD. ALL REFERENCES AVAILABLE IN THE 1D FORMAT